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ON

A STUDY OF CLINICAL PROFILE AND ELECTROCARDIO GRAPHIC CHANGES IN YELLOW OLEANDER SEED POISONING

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CERTIFICATE

*This is to certify that this dissertation entitled "**A STUDY ON CLINICAL PROFILE AND ELECTROCARDIO GRAPHIC CHANGES IN YELLOW OLEANDER SEED POISONING**" is the bonafide record work done by Dr.E.BOBBY, submitted as partial fulfillment for the requirements of M.D. Degree Examinations, General Medicine (Branch I) to be held in March 2008.*

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CONTENTS

INTRODUCTION

DELIBERATE Self harm[48] is an important problem in the developing world. Ingestion of yellow oleander seeds in Thanjavur district is very common due to its easy availability. It is being consumed more by the younger age group people.

Cardiac glycosides in the seeds cause vomiting, palpitation, cardiac arrythmias affecting the sinus and AV nodes and thereby causing mortality.

It was decided to study the yellow oleander poisoning in our place regarding the clinical profile factors deciding the mortality and modification of which would improve the prognosis in our patients.

OLEANDER IN VARIOUS SYSTEMS OF MEDICINE

Oleander, One of the common plants found abundantly itself in South India, is believed to have its origin in India[70]. It is also found in Tropical America Southern United States Hawaii and Srilanka.

Charaka (1000 BC) a pioneer in Ayurvedic medicine and Sushrutha (800 BC) the ancient surgeon have made references in their respective treatises about this plant.

According to literature in ancient Indian medicine including AYURVEDIC, SIDDHA, UNANI and OTHER INDIGENOUS SYSTEMS OF MEDICINE Oleander has been used as a medicinal plant to treat various disease conditions.

USAGE IN **AYURVEDIC SYSTEM OF MEDICINE**

Oleander plant was known in ancient India since Vedic age. Valmiki's Ramayana, the ancient epic, mentions about this plant[71].

Oleander was used as an external application for many skin conditions, and also as an internal medicament for dropsy. Rheumatism was treated with a mixture of Oleander leaves with curd[10]. Other uses were as an antiseptic and treatment for chest disease with cough[56]. It was

used for anal fissure and hemorrhoids[70]. The whole fruit was used as an abortifacient[30,47,65,66]. We find its mention about usage as a purgative[10,47], emetic[47] and anti malarial in various references. It was used in the treatment of periodic fever and ureteric stone[10,64].

USAGE IN '**SIDDHA SYSTEM OF MEDICINE**'

In siddha system of medicine various preparations from yellow Oleander had been tried for many conditions[51].

This included fever, skin disorders, leprosy, anorexia, polydipsia, impetigo, Syphilis, Furunculosis and inflammatory disorders. A few drops of root oil mixed with gingelly oil had been used to treat toothache[47]. It was used as a germicidal also.

USAGE IN '**UNANI SYSTEM OF MEDICINE**'

Yellow Oleander was widely quoted in Unani system of medicine.

Paste made from the leaves was used in skin disorders, powder of the leaves have been tried externally for bruises and superficial wounds[70]. Syphilis and leprosy were treated by an extract from roots and leaves[10]. Paste of the root had been used as aphrodisic[47]. It was applied externally over the male sex organs to correct sexual disability and to improve virility.

USAGE IN ‘OTHER SYSTEM OF INDIAN MEDICINE’

In other systems of Indian medicine like traditional systems, religious systems and community systems, recipe from yellow Oleander was used to treat parasitic infestation, leprosy, itching of breast, improvement of vision, toothache and wound dressing, urethral discharge piles, leucoderma and bronchitis. The roots were made into plaster and applied over the tumour[12]. It was used in the treatment of heart failure and atrial fibrillation in 1930’s.

FOR SMOKING

Oleander leaves were used for smoking but certain quote excess smoke will be poison[70,28,56].

OLEANDER POISONING – A HISTORICAL REVIEW

OLEANDER POISONING – A HISTORICAL REVIEW

Oleander plant was known since ancient times, finds mention in CHARAKA SAMHITHA under 30-40 different names in Sanskrit language[70].

The name thevetia was given in honour to Mr.Andre Thevit who travelled extensively in South America during 16th century and who has written extensively about this plant[70].

Some feel PERU as an original place of Oleander and hence Thevetia Peruviana[64]. Various names of yellow Oleander are EXILE, CEREBRA THEVETIA, THEVETIA NERIFOLIA, PILA KANER[47,1,68].

All parts of the oleander plant including smoke from burning cuttings and water in which the flower is placed are poisonous[21,28].

The kernels of seeds[43,47,30] are eight times more poisonous than the leaves followed by latex[43,69] of the plant. Compared to other parts flower, bark, root and stem are less poisonous.

It was used for procuring criminal abortion[5,50,56,65,66], Suicidal and homicidal[51,61] purpose, love philter, poisoning cattle[9,56,] with the paste concealed in corn or chapathi.

These names include Ashwaghna, Ashwaha, Ahswadya and Aswantak means killer or enemy of horse[10].

Karavira is an another name in which kara means a sword used in battles[36,].

Shatakundu meaning shata as hundred and kundu a hammer brings out its dangerous effects clearly[70].

Other names used were Viraka meaning a very potent fierce nature, Lakuda meaning a weapon made of stick, Rakthapuspha meaning a blood flower depicts its toxic or poisonous nature.

From the above names we clearly understand that different parts of Oleander plant were used or rather abused since ancient times because of its Lethal qualities.

Apart from its role as a cattle poison, the fruit and kernel were used to kill fish and insect[56].

Arrows were poisoned with Oleander extract by South American and East African Tribes.

Pigs and Jackals were poisoned with Oleander extract by Mundas a Himalayan Tribes.

Reports about numerous deaths from Oleander branches being used as a food skewers, from Oleander smoke and from Oleander flower were of historical interest as well as present and future interest[28].

PHOTOGRAPH 1 :
YELLOW OLEANDER PLANT

BOTANICAL DETAILS

Oleander is being cultivated and scattered all over India. Thevetia Nerifolia Juss is an ornamental plant with cosmopolitan distribution.

It grows as a hedge plant or erect brush or as a small tree[47,50]. It grows quickly and becomes a wild plant covering a large area. It grows quickly from seeds and cutting transplants well and has a tendency to ensure drought and frost to some extent[24,57].

PLANT CLASSIFICATION[44]

CLASS	-	Dicotyle donae
SUB-CLASS	-	Gamopetalae
SERIES	-	Bicarpellatae
COHORT	-	Gentianales
FAMILY	-	Apocyanaceae

SHRUB OR TREE

Looking attractive and ever blooming, this small tree grows to a height of 12-15 feet and 8-10 inches thickness. Complete naturalization has occurred in India. This is shown in photograph 1.

PHOTOGRAPH 2: A STEM AND LATEX OF YELLOW OLEANDER

PARTS OF THE PLANT

STEM

It is solid, cylindrical, erect and branched and has a milky latex[4,22]. This is shown in photograph 2.

LEAVES[41,47]

Oleander leaves are 5 inches long. It can be sub-sessile, sessile alternatively spiral or irregularly scattered on the stem. Lanceolate or linear in shape. The surface of the leaf is shiny. Entire leaf margin or only the tip may have a pinnately reticulation and venelation. Bright green in colour and an acute apex are other features. This is shown in photograph 3 & 3A.

FLOWERS

In spite of its poisonous nature, the waxy yellow flowers of the Oleander has its own fragrance it is short lived[54,16]. The flowers are bell shaped twisted spirally with 5 lobes[47]. It measures 5-7cm in length and 5cm in breath. The flowers may exhibit a pale buff cream coloured or coppery appearance some times. Blossoms continue to appear throught the year[24]. This is shown in photograph 4 & 4A.

PHOTOGRAPH 3:
LEAF OF YELLOW OLEANDER

FRUIT

It is globular in shape and light green in colour. It's size is 4-5cm diameter and contains a single nut[34,47]. This is shown in photograph 5 & 5A. Lethal dose in children is about one to two fruits[47,53,55].

NUT

It is triangular or odd shaped. It has a deep groove along the edge. It is light brown colour. Each nut contains two seeds. It is hard like a stone. This is shown in photograph 6 & 6A. 8 to 10 seeds are fatal in adults.

SEED

It is pale yellow in colour and hard like a stone, it has a covering, kernel and content of glycosides[70,51,]. 2 to 5 seeds are present in a single nut. This is shown is photograph 6 & 6A.

LACTIFEROUS TISSUE[24]

It consists of thin walled greatly elongated much branched ducts containing milky fluid called Latex. Lactiferous ducts are two types, Latex vessels and Latex cells. They have numerous nuclei which lie embedded in this layer of protoplasm lining the cell wall which is usually thin and made up of cellulose.

PHOTOGRAPH 4 :
FLOWER OF YELLOW OLEANDER

LATEX[4,24]

It is a milky juice secreted by the plant, It always contain some waste products and it is often irritating and poisonous. It causes inflammation and even blister when it comes and contact with skin. This Latex is present in Latex cells. The secretions of this milky juice is for defence purpose and animals avoid such plants. This is shown in photograph 2.

LATEX COMPOSITION

Latex occurs as an emulsion and consisting of variety of chemical substances. Among the nutritive materials sugar, starch grains, proteins and oils are often found. The waste products in Latex include gum, resin, tannin, alkaloids, rubber etc. Latex also contains some salts, enzymes and poisonous substances.

The exact function of Latex is not clear perhaps in someway it is associated with nutrition, healing of wounds and protection against parasites and animals.

PHOTOGRAPH 5 :
FRUIT

PHOTOGRAPH 5A:
FRUITS AND FLOWER OF YELLOW OLEANDER

PHOTOGRAPH 6:
A TRIANGULAR NUT WITH SEEDS

PHOTOGRAPH 6A:
A TRAINGULAR NUT WITH SEEDS

CHEMICAL CONTENTS – ALKALOIDS[24,70]

Alkaloids are combination of organic acids and complex nitrogenous substances[38]. They may be by products of nitrogen metabolism in plants. They have an intensively bitter taste and are extremely poisonous. Majority of them are crystalline solids which are insoluble or sparingly soluble in water but readily soluble in alcohol. Alkaloids are generally but not universally formed in the roots and from there translocated to certains organs in the plant. The role played by the alkaloids in the physiology of plant cells are not known.

The most toxic glycosides[56,30] in yellow Oleander are 1) Peruvoside 2) Ruvoside 3) Thevetin – A, 4) Nerifolin, 5) Cerebrin 6) Thevetin – B, Peruvoside, Thevetin A & B produce effects similar to digitalis, but have a more rapid onset of action.

Yellow oleander does not lose its toxicity even when dried. Glycosides in yellow oleander undergo extensive enterohepatic cycling[56].

Amount of glycosides per plant part is 0.07% in leaves. 0.045% in fruit, 4.8% seed kernel, 0.036% in sap[56].

Thevetin cross the placenta[56].

OLEANDER : ITS VARIOUS ALKALOIDS AND CHEMICAL STRUCTURES

OLEANDER : ITS VARIOUS ALKALOIDS AND CHEMICAL STRUCTURES

Oleander (thevetia nerifolia) contains many glycosides which are mainly of cardiac action[33,63].

This cardiac active glycosides have two major constituents[60,69]

i. Aglycone or Genin or Non-sugar compound.

ii. Sugar compound.

AGLYCONE OR GENIN

This aglycone is essential for the cardiotonic and pharmacological activities. It has a steroid structure to which either five or six membered unsaturated lactone ring is attached at c_{17} position of steroid nucleus[69]. These aglycones are broadly divided into two sub groups. That is,

i. Cardenolides

ii. Bufadenolides

Most of the aglycones of thevetia nerifolia belongs to cardenolide group.

SUGAR COMPOUND[3,60]

These molecules are attached to aglycone. These sugars determine the water solubility, cell permeability and potency of glycosides. The duration of action of glycosides depends on the type and number of sugar molecules attached to it. The sugar chain consists of 1 to 5 sugars

occurring either as a single or repetitive units. Some of the unique sugars in cardiac chemistry include

DIGITOXOSE, 6 – DEOXY SUGAR, 2 – DEOXY SUGAR, 2,6 – DEOXY SUGAR, DIGITALOSE, GENTIOBIOSE AND THEVETOSE.

Gentibiose and thevetose are more important sugars in cardiac glycosides of thevetia nerifolia. In the process of isolation of glycosides, So far monoside of digitoxigenin its 19-oxo form cannogenin and its 19-oxy form cannogenol have been derived from the seeds and kernels of Thevetta nerifolia.

The cardenolide type of cardiac glycosides in yellow oleander plant are found in maximum quantities in the seed and kernels[69]. Root, stem, bark and leaves contain lesser quantites[54]. The cardenolide glycosides are further broadly divided into two groups[11,34] they are –

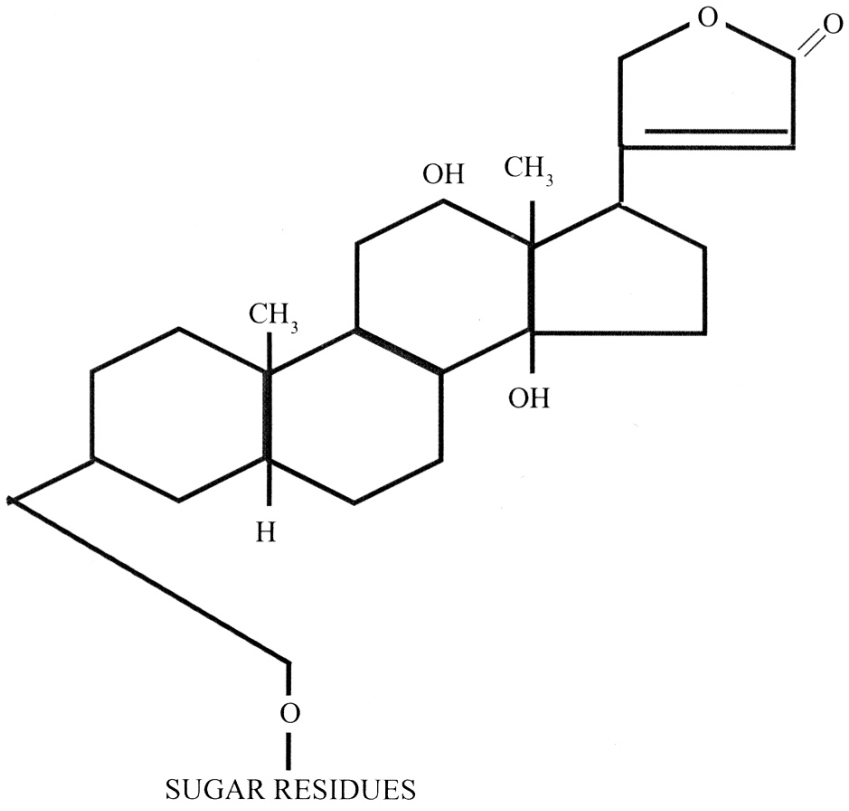
i. Polarglycosides – Trioside

ii. Secondary glycosides – Monoside

The POLARGLYCOSIDES include thevetin-A, thevetin-B, acetyl thevetin-B and thevebioside.

The secondary glycosides include nerifolin, cereberin, peruvoside, ruvoside, thevernerin, thevefolin, peruvoside monoacetate and substances A,B,C & D.

The important cardiac glycosides are discussed in detail.



Chemical Structure of **DIGOXIN**

THEVETIN-A[3,25]

It has one methoxyl group and sugar. The sugar content is L.Thevetose plus two glucose. The chemical structure is shown in figure–1.

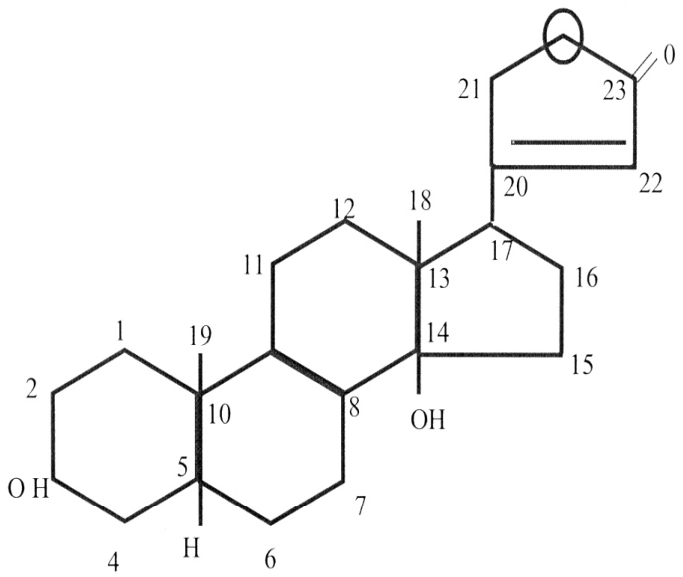
PHARMACOLOGICAL ACTIONS[70]

Small doses of thevetin-A has stimulating effect on heart while large doses could depress the heart and could arrest ventricular contraction. Reports from many studies showed when the heart was stimulated by small doses of Thevetin, there was an increase in cardiac output and coronary blood flow. Large doses of thevetin would reduce the coronary blood flow.

EFFECTS ON HUMAN HEART[13]

Administering 1 to 5 cat units of Thevetin-A by oral route will cause definite reduction in heart rate. The maximal effect of the drug was apparent in 2-3hours. Thevetin A was found to be slowly and irregularly absorbed by gastrointestinal tract. IV infusion produces full effect in about 6 minutes and the effect disappearing in about 2-3 hours.

The effect of thevetin-A on blood pressure is equivocal. When thevetin was given to patients with congestive cardiac failure, it improved the cardiac failure by reducing the venous pressure and by slowing cardiac rate. Atrial Fibrillation showed improvement with thevetin.



Basic chemical structure of **AGLYCONE or GENIN** with lactone ring in position 17, tertiary hydroxyl in position 14 and secondary hydroxyl in position 3.

ELECTROCARDIOGRAPHIC CHANGES[47,56,29,46,52]

Electrocardiographic changes noted with thevetin are

BRADYCARDIA,

T WAVE INVERSION,

PROLONGED PR-INTERVAL,

AV DISSOCIATION,

VENTRICULAR TACHYCARDIA

VENTRICULAR FIBRILLATION.

Thevetin was also useful in reducing tachycardia of hyperthyroidism. Thevetin was used successfully in post operative thyroid crisis.

THEVETIN – B[3]

[Cerebroside]

It has one methoxyl group and the sugar content is L-thevetose plus two glucose. The chemical structure is shown in figure2.

PHARMACOLOGICAL ACTIONS[70]

It has cardiotonic activites. It has very weak digitalis like action and it is one of the weakest of thevetia glycosides with the cardiotonic effects. The ECG changes are similar to digitalis toxicity.

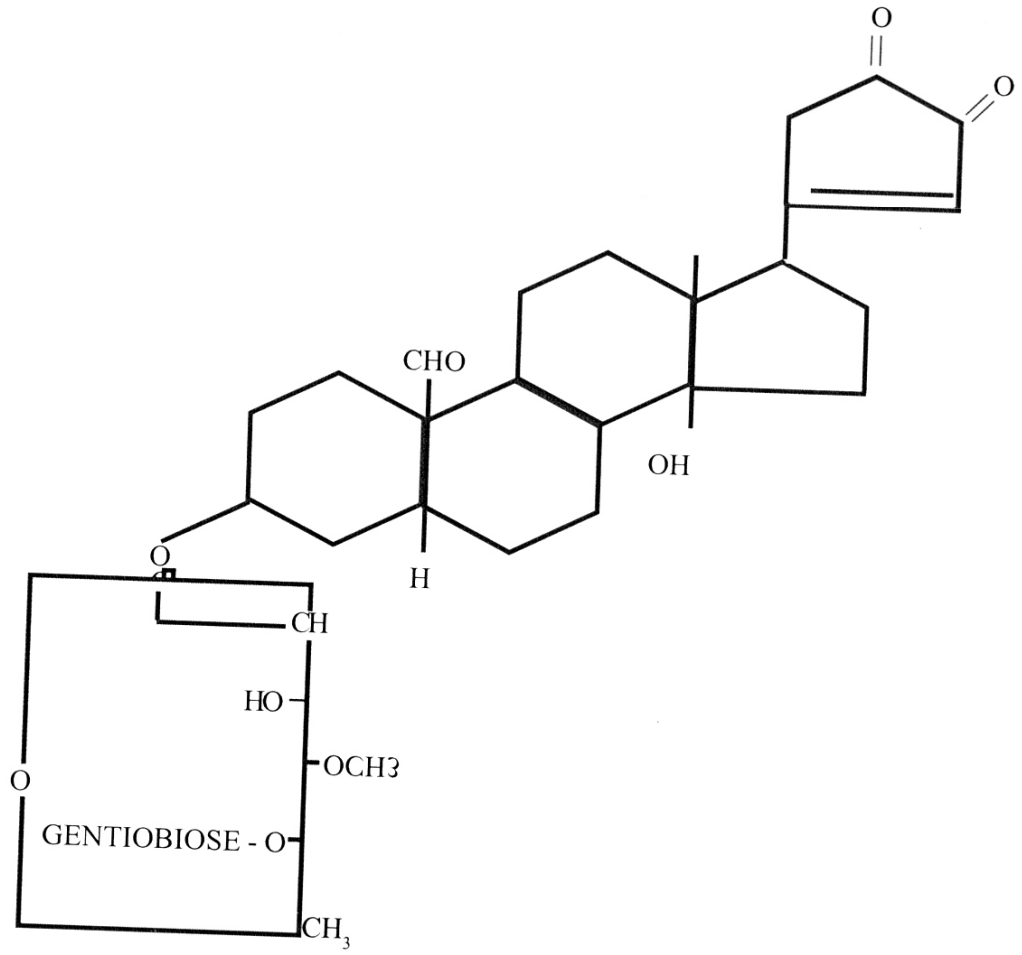


Figure 1 : Chemical Structure of **THEVETIN - A**

ACETYL THEVETIN – B

It has one methoxyl and one acetoxyl unit. Deacetylation with potassium bi carbonate gives thevetin-B. The chemical structure is shown in figure3.

NERIFOLIN[70]

This is the major monoside of Thevetia. It has one methoxyl unit, and L-Thevetose forms the sugar unit. The chemical structure is shown in figure4.

PHARMACOLOGICAL ACTIONS[30,31]

A moderately potent cardiac glycoside having cardiotonic activities.

CEREBERIN[3]

(Monoacetyl Neriifolin)

It has one methoxy and acetoxyl groups. It is practically insoluble in water. The chemical structure is shown in figure5.

PHARMACOLOGICAL ACTIONS

Cereberin has strychnine like activity. It has cardiotonic activity.

In experimental animals, cereberin produces systolic cardiac arrest, rise in Blood Pressure, arrhythmias and circulatory collapse.

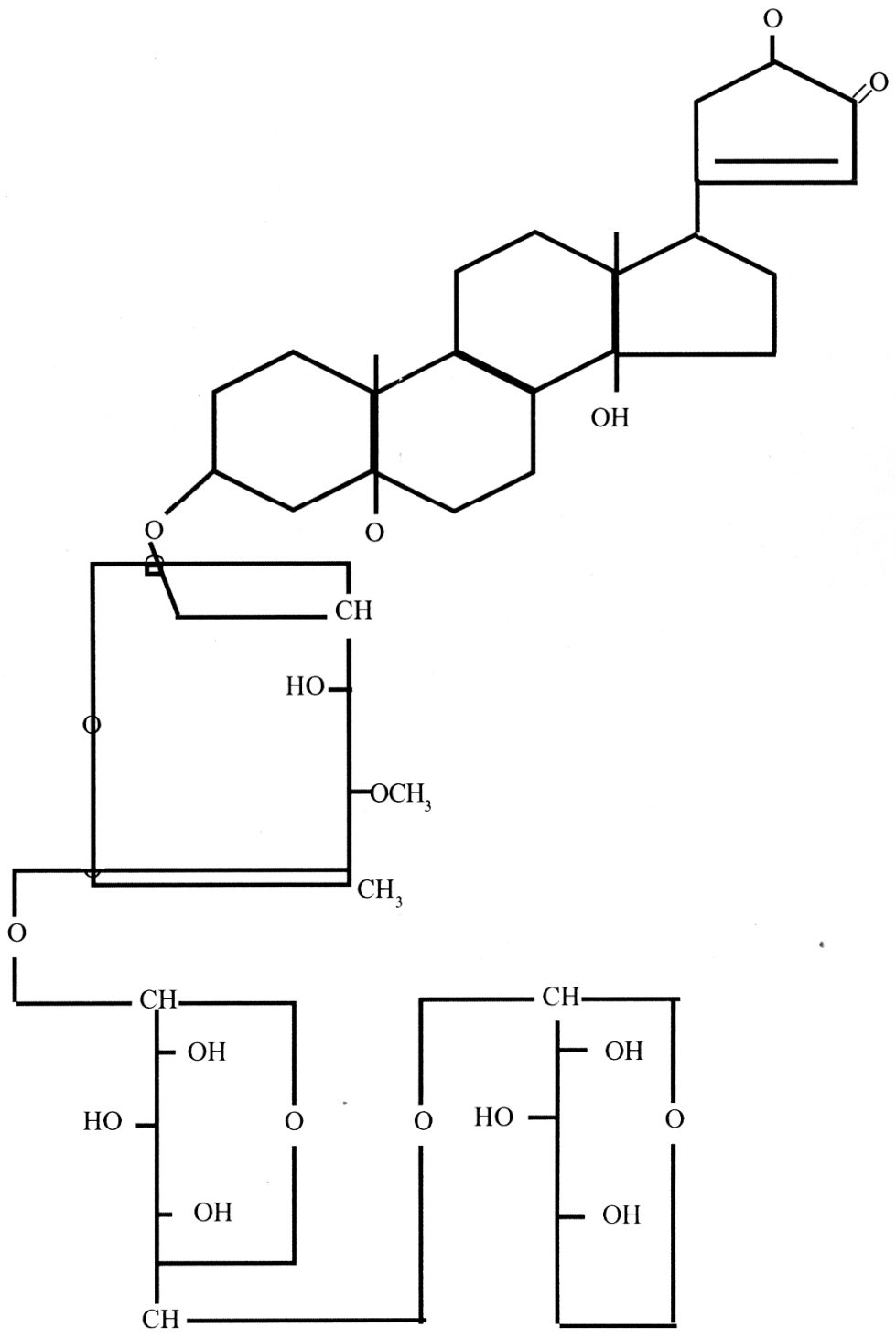


Figure 2: Chemical Structure of **THEVETIN - B**

PERUVOSIDE[3,7]

(Oxo-Nerifolin)

It has one methoxyl group and L-thevetose molecule. Peruvoside contains an aldehyde group. Peruvoside is more prone to auto-oxidation especially in liquid form. The chemical structure is shown in figure6.

PAHRMACOLOGICAL EFFECTS

Many studies have been done for the effects of peruvoside on cardiovascular system[47]. Peruvoside is effective by oral and intravenous routes.

Duration of action of the peruvoside is short. It has low serum protein binding nature just like OUABAIN[3]. Its effect on the serum in therapeutic and toxic doses closely resembles the other cardiac glycosides.

This glycoside is absorbed more from the stomach and exertion is mainly by bile, urine and faeces. A single dose of peruvoside disappeared completely in 72-96 hours. There was evidence for the presence of enterohepatic circulation[56]. The elimination of peruvoside was faster than digitoxin[70].

The cardiotonic and haemodynamic effects of peruvoside on failing heart was done extensively by many workers[3,7,49]. They observed the

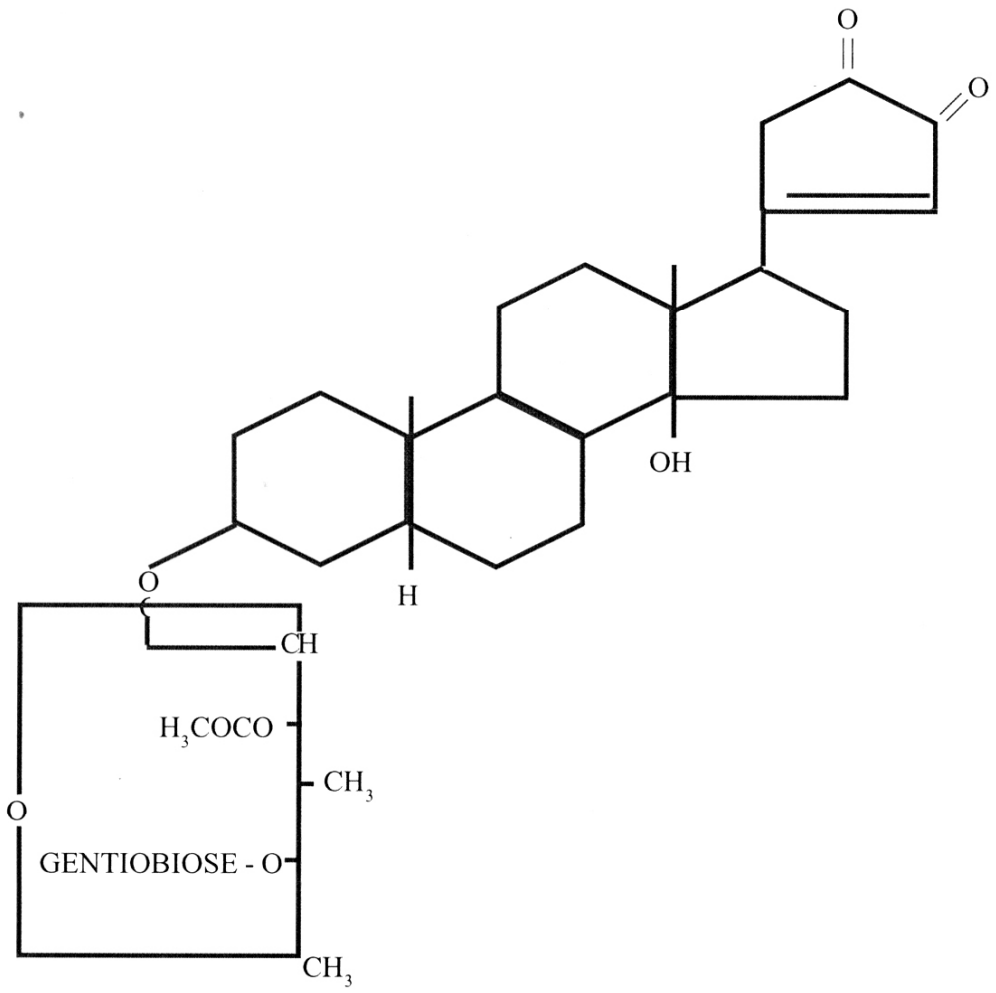


Figure 3 : Chemical Structure of **ACETYL THEYETIN - B**

following hemodynamic effects of peruvoside in congestive cardiac failure.

PARAMETER	EFFECT
Force of contraction	- Powerful positive ionotrophic
Heart rate	- Decreased towards the normal heart rate
Cardiac output	- Increased
Right atrial pressure	- Decreased
Left ventricular pressure	- Increase in sytole and mild fall in diastole
Systolic Blood pressure	- Increased
Cardiac minute volume	- Increased
Total peripheral resistance	- Elevated Coronary blood flow
Coronory blood flow	- Augmented
Conductivity and excitability	- Unaffected

EFFECTS OF PERUVOSIDE ON NORMAL HUMAN HEART

The following changes were found after intravenous peruvoside[13].

1. Increase in ejection velocity in early systole.
2. Shortening of pre-ejection period.

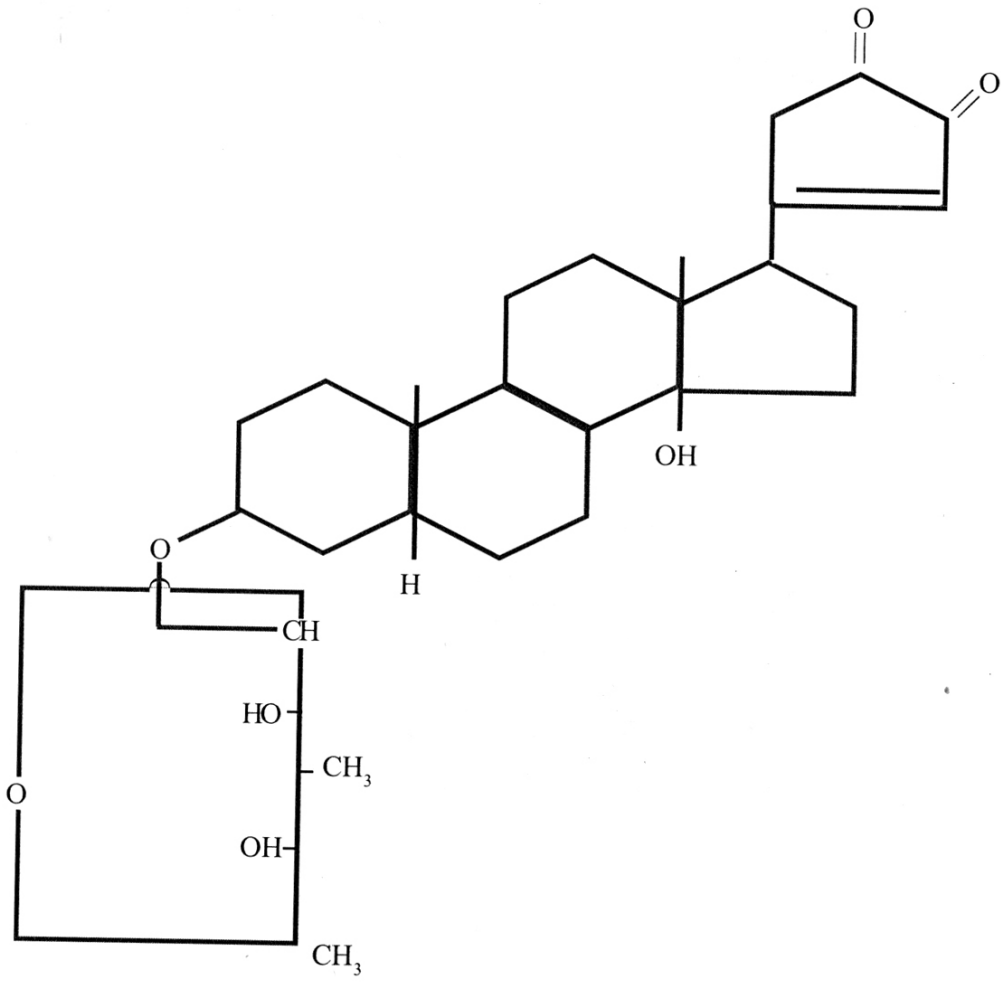


Figure 4: Chemical structure of **NERIFOLIN**

3. Shortening of total systolic time with left ventricular ejection time remaining constant.
4. Decrease in heart rate.
5. Increase in systolic blood pressure with diastolic pressure remains constant.
6. Flattening of 'T' waves in ECG.

The effects of peruvoside on congestive cardiac failure were studied by Bhatia et al 1970. They observed the following effects.

1. Increase in cardiac index.
2. Increase in stroke power
3. Decrease in pulmonary arterial pressure.

Thus peruvoside improved the myocardial function at a lowered left atrial pressure.

SPECIAL INDICATIONS for peruvoside therapy were cardiac Insufficiency with bradycardia, latent cardiac insufficiency, insufficiency of senile heart and corpulmonale chronicum and in the prophylactic treatment of serious conditions with stress, like surgery[70].

Advantages of peruvoside over ouabain were its suitability for both oral and intravenous administration, useful in treating acute and chronic cardiac failure and small alterations in ECG compared to other glycosides. Peruvoside had low toxicity than other glycosides.

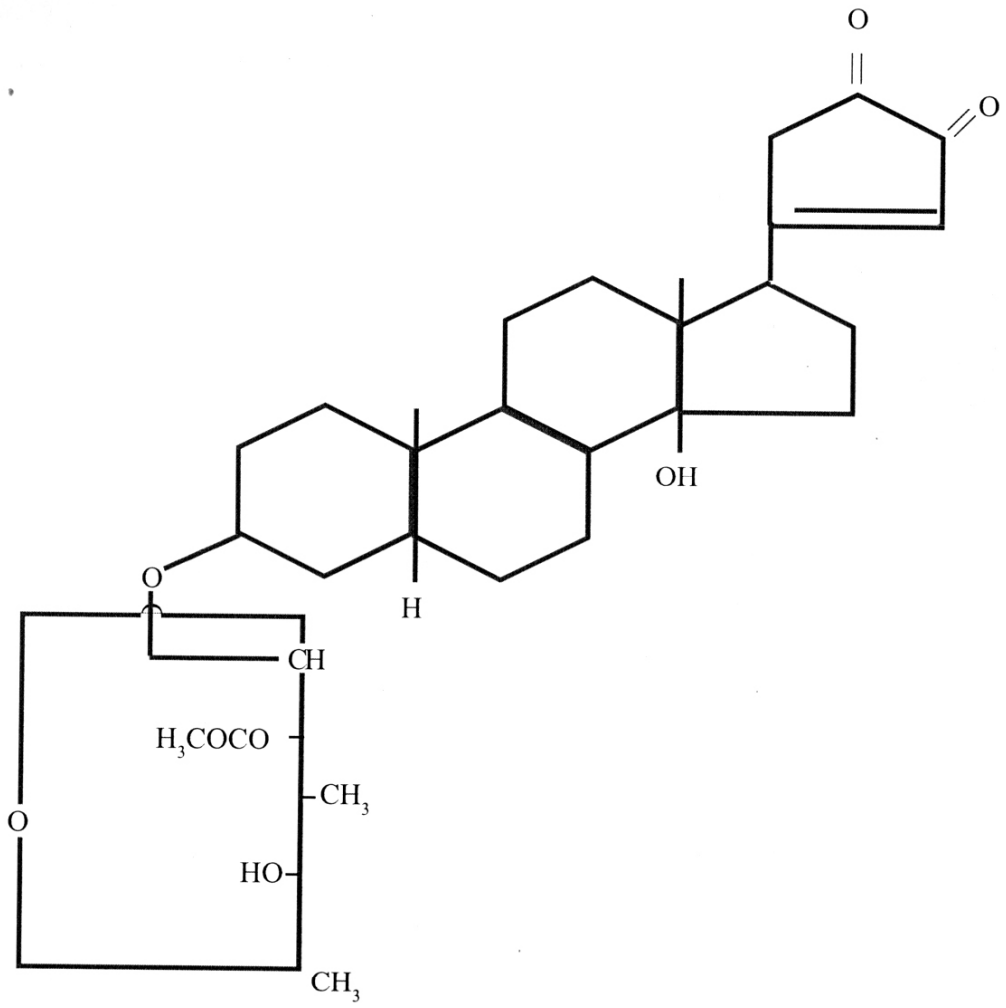


Figure 5 : Chemical Structure of **CEREBERIN**

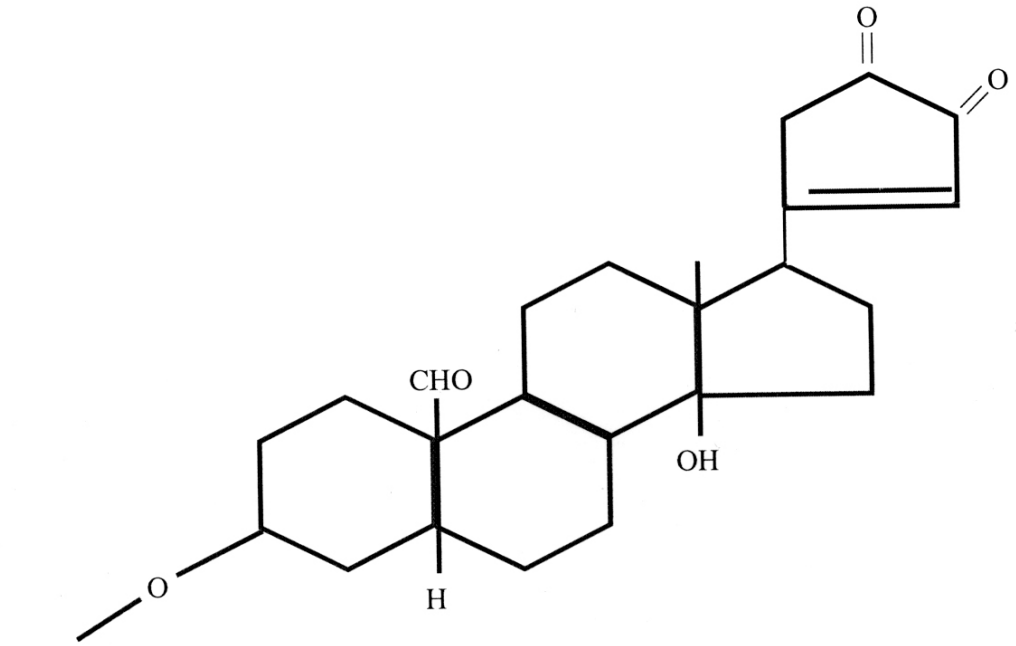


Figure 6 : Chemical structure of **PERUVOSIDE**

RUVOSIDE[72]

(Thevenerin)

It has one methoxyl group and L-thevetose forms the sugar moiety. The chemical structure is shown in figure –7.

PHARMACOLOGICAL ACTIONS[70]

The absorbtion range of ruvoside is 7 to 27% from the intestinal tract and completely eliminated from body in 72 to 96 hours. Ruvoside is quick and short acting glycoside with cumulative toxicity[3].

At lower concentration, it produced a marked increase in the force of contraction accompanied by slowing of heart rate. In higher concentration, the heart stopped before any evidence of the positive ionotrophic effect could be noted. Ruvoside has a powerful emetic effect.

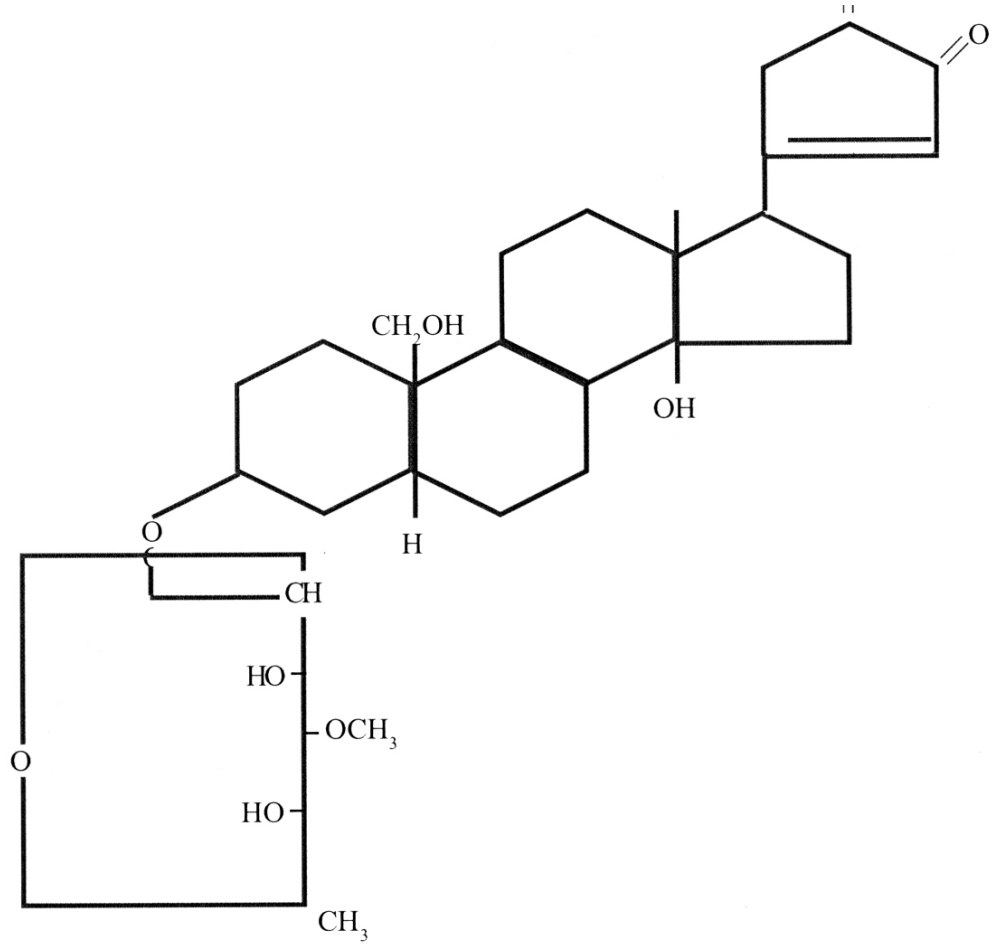
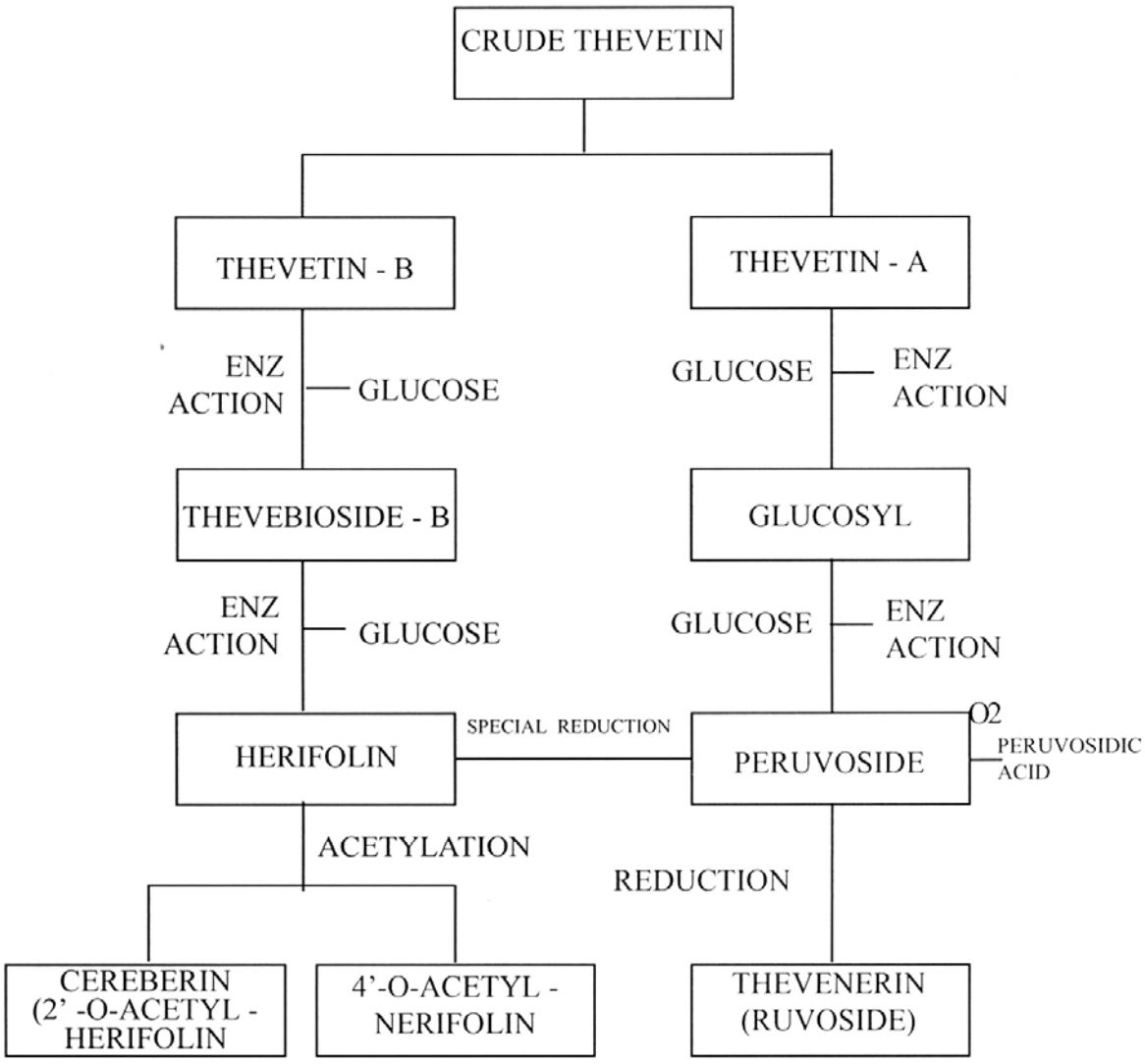


Figure 7: Chemical Structure of **RUVOSIIDE**

CHEMICAL INTERRELATIONSHIP OF THEVETIA GLYCOSIDES



POSSIBLE MECHANISMS OF ECG MANIFESTATIONS IN YELLOW OLEANDER POISONING

POSSIBLE MECHANISMS OF ECG MANIFESTATIONS IN YELLOW OLEANDER POISONING.

Yellow Oleander seed poisoning causes a wide variety of rhythm disturbances, electrocardiographic variations and abnormalities[47,56]. Some of them primarily due to cardioactive glycosides[23] and some are secondary to other factors like electrolyte imbalance, hypoxia, acidosis etc[26].

Cardiac glycosides in yellow oleander will alter the cardiac muscle physiological properties[69] such as automaticity, rhythmicity, conductivity and contractility.

AUTOMATICITY AND RHYTHMICITY[32,35,45]

Automaticity and rhythmicity are the primary function of Sino Atrial Node which has the most automatic cells and hence it is the natural pacemaker. Clinically arrhythmias may result from alteration of automaticity of sinus node or from an abnormal impulse formation from ectopic automatic tissue.

In yellow oleander poisoning the abnormality in automaticity and rhythmicity are seen as follows.

A. SLOWING OF AUTOMATICITY – Sinus bradycardia

B. ALTERATION OF RHYTHMICITY – Escape rhythm formation presenting as.

1. Sinus bradycardia with juctional escape rhythm.
2. AV dissociation
3. Escape rhythm without sinus activity.

With the onset of escape rhthym there is a shift in the pacemaker activity and the subsidary pacemaker which remain latent under normal condition because of faster rate of impulse formation by SA node, becomes active. In oleander poisoning AV node takes the brunt and becomes the secondary pacemaker in many instances, which may present as

1. AV nodal tachycardia
2. Tachy-Brady syndrome.

Abnormalities of depolarization and repolarization in yellow oleander poisoning are represented by ST depression and T wave inversion.

Many factors influence the repolarization process. Some of them in oleander posioning are myocarditis and electrolyte imbalance.

In yellow oleander poisoning most of the primary T wave changes seen are suggesting an abnormality of the repolarization due to toxic effects, producing an element of myocarditis and furthur influenced by electrolyte imbalance mostly related to potassium changes[56].

ANATOMICAL BASIS OF ECG CHANGES IN YELLOW OLEANDER POISONING

From various ECG changes it is possible to isolate certain areas in conducting system which are affected in yellow oleander poisoning.

SINO ATRIAL NODE

SA node is commonly involved[64] and has been shown in the form of DEPRESSION and also as a part of SICK SINUS SYNDROME. The brady type of sick sinus syndrome is most common type.

ATRIO VENTRICULAR JUNCTION

It is also commonly affected by toxic principles of oleander and this leads to variety of rhthym disturbances[69] like A.V.nodal rhythm, AV nodal tachycardia etc.

BUNDLE OF HIS

Right bundle branch is occasionally involved.

Left bundle branch is never involved.

Intraventricular portion is rarely involved.

MYOCARDIUM

Myocardium is involved in different ways eg: myocarditis made out by various non specific changes producing ST and T wave changes.

ECG abnormalities in yellow oleander poisoning is multifactorial. So it can be discussed in relation to various factors.

1. The role of cardioactive glycosides.
2. Contributory Autonomic system alterations in arrhythmogenesis.
3. Electrolyte imbalance
4. Myocarditis
5. Acidosis
6. Hypoxia
7. Hypotension and shock
8. Vascular factors
9. Combination of factors.

ROLE OF CARDIO-ACTIVE GLYCOSIDES.

Thevetia nerifolia contains cardioactive glycosides. Yellow oleander glycosides closely resemble other cardiac glycosides in structure as well as toxic and pharmacological effects. In decreasing order of potency the glycosides of yellow oleander are Peruvoside, Ruvoside, Thevetin-A, Neriifolin, Cereberein and Thevetin – B[56]. They are all have an aglycone (digitonigenin or its derivatives) combined with a sugar and act by inhibiting NA+K+-ATP ase[31,67].

SODIUM POTASSIUM –ATP ASE

In 1957, Jens Skou discovered an enzyme that hydrolysis ATP only if Na+ and k+ are present in addition to Mg2+ which is required by all ATP ases and names as Na+k+-ATP ase.

Cardiotonic steroids inhibit the pump and the ATP ase, only when they are located outside the cell. The Na+K+ATP ase is an integral membrane protein with two subunits with the molecular weight[2] of ~50,000 and ~1,10,000.

The activity of this Na+K+ATP ase is an essential cell function. Every animal cell maintains a low concentration of Na+ and a high concentration of k+ than is found in its surrounding medium. This is done by Na+k+-ATP ase which couples breakdown of ATP to simultaneous movement of both Na+ and k+ against their concentration gradient. For each molecule of ATP converted to ADP and pi, this transporter moves 2k+ions inward and 3 Na+ions outward across the plasma membrane and this process is electrogenic.

INHIBITION OF NA+K+ATP ASE[6,17,25,31]

All glycosides in yellow oleander are potent inhibitors of active transport of Na+ and k+ across cell membrane by binding to a specific site on the extracytoplasmic face of α subunit of Na+k+ATP ase.

The binding of cardiac glycosides to Na+k+-ATP ase and inhibition of cellular Na+ pump is reversible and entropically driven. These glycosides bind preferentially to the enzyme following phosphorylation at β-asparate on the cytoplasmic face of the ∝ subunit and stabilize this conformation known as E2P.

The positive inotropic effects of the glycosides are due to an increase in the availability of cytosolic Ca++ during systole to interact with contractile proteins to increase the velocity and the extent of sacromere shortening. This increase in intracellular Ca++ is one of consequences of cardiac glycoside induced decreased cycling of the sarcolemmal Na+k+ATP ase.

Hence it can be definitely concluded that some of the ECG changes are due to the presence of cardioactive glycosides and principle in yellow oleander, which are toxic to conductive system.

CONTRIBUTARY AUTONOMIC NERVOUS SYSTEM

Though the role of autonomic nervous system is not clear, it appears that it contributes to the ECG abnormalities.

Increased vagal tone is responsible for the following ECG changes like sinus bradycardia, sinus arrhythmias, SA block, premature beat, AV

block, junctional rhythm, junctional escape, AV dissociation, ventricular escape and cardiac arrest.

Yellow oleander seeds contain many cardioactive glycosides and some of them are similar to digitalis and this can be explained on the basis of similarities in basic chemical structure and sugar linkage. Many of the above rhthym disturbances are therefore probably induced by autonomic imbalance mediated in many cases. Hence Atropine can be used as a diagnostic agent to unmask autonomic imbalance in arrhythmogenesis induced by toxic principles in yellow oleander seeds.

ROLE OF ELECTROLYTE IMBALANCE

Electrolyte imbalance are commonly seen in yellow oleander poisoning. The biochemical features include hyponatremia, hyperkalaemia and hypochloremic acidosis. Hyperkalaemia is commonly associated with yellow oleander poisoning. The potassium abnormalities are also contributing to arrhythmias in yellow oleander poisoning.

ROLE OF MYCARDITIS[52]

Yellow oleander poisoning may produce a picture of toxic myocarditis and this can explain some of the ECG changes.

Although there is no single specific ECG abnormality diagnostic of myocarditis, most common ECG finding is diffuse 'T' wave change in the form of T wave inversion in many leads. The other changes are varying degrees of AV Block, Bundle branch block, non-specific intraventricular conduction disorders, ectopics etc. The uncommon features are non-specific ST-T changes in the form of sagging or depression, prolonged QT, notched (or) broad P and ectopic tacharrhythmias.

Hence an element of myocarditis is present in yellow oleander poisoning and steriods are used in the treatment.

ROLE OF VASCULAR FACTORS[11]

Myocardial ischaemia secondary to involvement of the coronary artery may be an additional operative factor in the production of some of the ECG changes.

ST depression is classically seen in inferior and anterior leads. Although the glycosides and electrolytes can produce similar changes, coronary vasospasm was thought in experimental studies. Toxic doses of thevetin and subsidiary glycosides in the nuts of thevetin nerifolia can produce vasoconstriction[64].

AIM OF THE STUDY

AIM OF THE STUDY

1. TO STUDY THE ELECTRO CARDIOGRAPHIC CHANGES IN YELLOW OLEANDER SEED POISONING.
2. TO STUDY THE COMMON TYPE OF ARRYTHMIAS OBSERVED.
3. TO STUDY THE AGE AND SEX INCIDENCE IN OLEANDER POISONING.
4. TO CORRELATE THE NUMBER OF SEEDS WITH THE SEVERITY OF ECG CHANGES.
5. TO CORRELATE THE FORM OF CONSUMPTION OF SEEDS WITH THE SEVERITY OF ECG CHANGES.
6. TO STUDY THE BIOCHEMICAL PROFILE IN OLEANDER SEED POISONING.
7. TO STUDY THE NECESSITY OF STOMACH WASH.
8. TO STUDY THE COMMON SYMPTOMS IN OLEANDER SEED POISONING.
9. TO STUDY THE TIME LAPSE OF ELECTROCARDIOGRAPHIC CHANGES BECOMING NORMAL.

MATERIALS AND METHODS OF STUDY

MATERIALS AND METHODS

OF STUDY

DESIGN

This is a single centre non-randomised prospective study is determining the clinical profile and electrocardiographic changes among 51 patients with yellow oleander seed poisoning.

STUDY PERIOD

Patients in and around Thanjavur District admitted for yellow oleander seed poisoning in Thanjavur Medical College Hospital during the period of one year between July 2006 to June 2007 were taken up for this study.

STUDY CENTRE

This study was carried out at Department of Medicine, Thanjavur Medical College Hospital, Thanjavur, South India.

All cases were examined in detail in the wards and findings were recorded in the proforma annexed herein. All cases were followed till their discharge or death. Post mortem was done in the expired patient and the heart was studied in detail.

HISTORY DETAILS

All cases were examined for the age, sex, socioeconomic status and literacy.

All details about poisoning like colour of the oleander, number of seeds, form of consumption like paste, grounded or chewed, with or without leaves, with or without food or empty stomach were enquired.

Time of poisoning, time of admission to Hospital, episodes of vomiting, symptoms like chest pain, palpitation, giddiness, syncope, etc. details about first aid were enquired.

CLINICAL EXAMINATION

All cases were examined clinically for pulse rate, Blood pressure, State of Higher functions, cardio vascular system, respiratory system, central Nervous system and Gastro intestinal system involvement.

LABORATORY INVESTIGATIONS

URINARY EXAMINATIONS

Urine Albumin

Urine Sugar

Urine Deposits

BLOOD EXAMINATION

Blood Sugar

Blood Urea

Serum Creatinine

Serum Electrolytes

ELECTRO CARDIOGRAM

ECG was taken in all cases immediately after admission. Routine conventional limb leads, chest leads and long strip were recorded. Serial ECG were recorded at 12 hours interval for first 3 days of admission and thereafter ECGs were taken every 24hrs till discharge.

ECGs were interpreted fully in the following way as rate, rhythm, axis, 'P' waves, PR interval, QRS, T, ST.Q.Tc interval and 'U' Wave.

Positive observations in the ECG are shown here. The normal values of all ECGs are depicted in the ECG master chart.

AUTOPSY

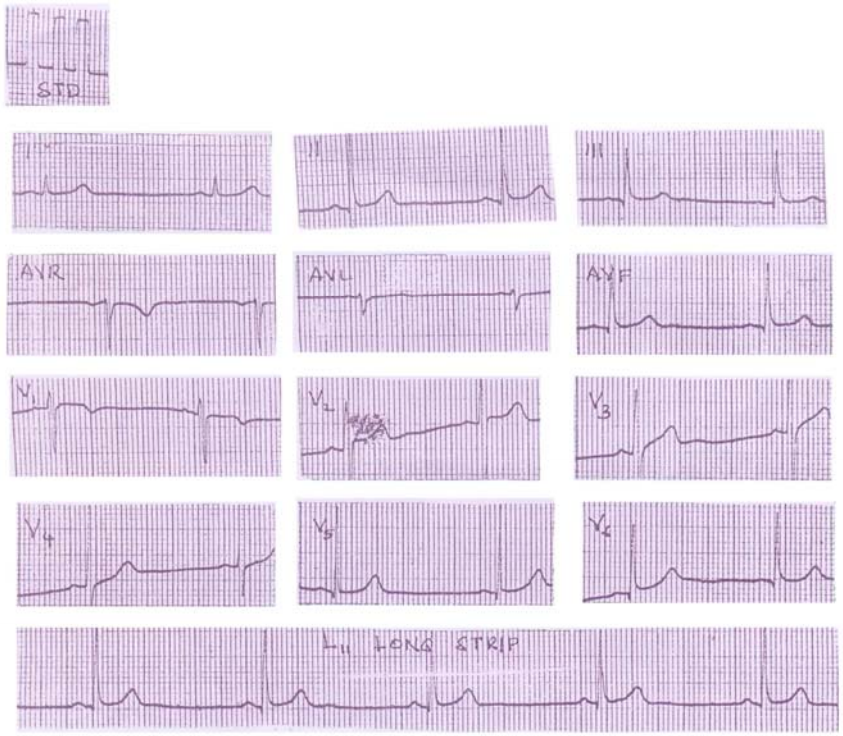
Postmortem was done in death. Macroscopic appearance of various organs and microscopic appearance of heart were studied in detail.

TREATMENT

Patients were treated with stomach wash, Few patients were treated with activated charcoal stomach wash, followed by Ryle's tube aspiration, IV fluids and steroids, with continuous monitoring of few cases. Atropine and orciprenaline were given if heart rate was below 60 per minute. Due to lack of pacing facilities temporary or permanent pacemaker was not tried.

CASE NUMBER - 17

ECG NUMBER - III



Electrocardiogram showing SINUS BRADYCARDIA Evidenced by RR interval of 1.28 seconds, a rate of 47/minute with normal P,QRS - T complexes

RESULTS, OBSERVATIONS AND DISCUSSION

RESULTS, OBSERVATIONS

AND DISCUSSION

AGE INCIDENCE

Out of the total of 51 cases, the number of patients between the age group of below 20 were 20(41.17%) and between the age group of 21-30 were 19(37.25%) and between the age group of 31-40 were 11(21.56%) and in the age group of above 70, 1 patient(1.96%).

Incidence of poisoning is more in age group below 20 years. It is shown in table 1 given below.

Tab 1. Showing Age incidence

AGE GROUP IN YEARS	NUMBER OF PATIENTS	PERCENTAGE
20 and below	20	41.17%
21 – 30	19	37.25%
31 – 40	11	21.56%
Above 70	1	1.96%

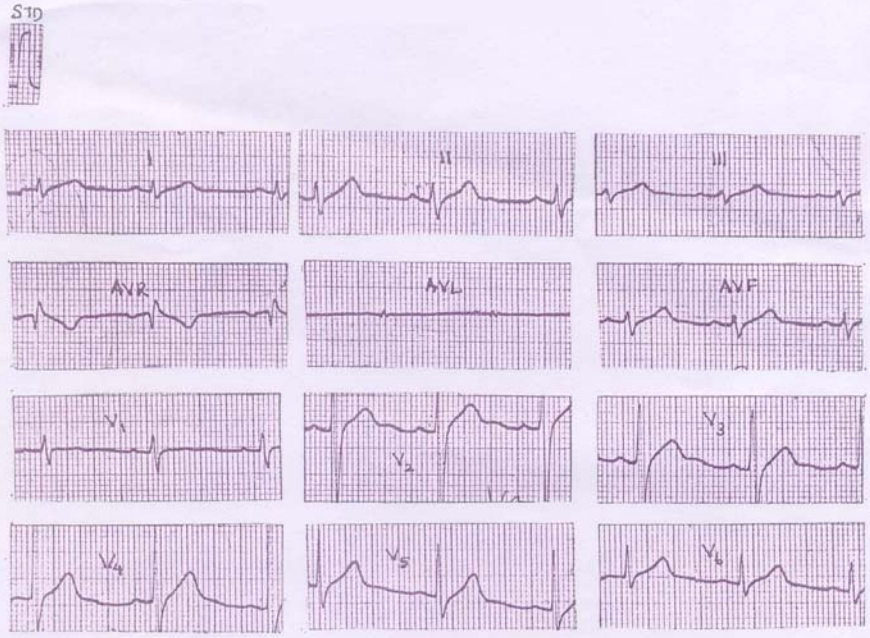
SEX INCIDENCE

Out of the total of 51 cases, 19 cases were males and 32 were female.

Male : Female ratio was 1:1.6.

CASE NUMBER -1

ECG NUMBER - VIII



NORMAL ELECTROCARDIOGRAM - RR interval of 0.88 seconds, rate of 68/minute, PR interval of 0.16 seconds with normal P,QRS - T complexes.

Percentage of males in this study was 37.2%. In men the common age group was 21-30 years and the number of cases in this age group was 9(47.36%).

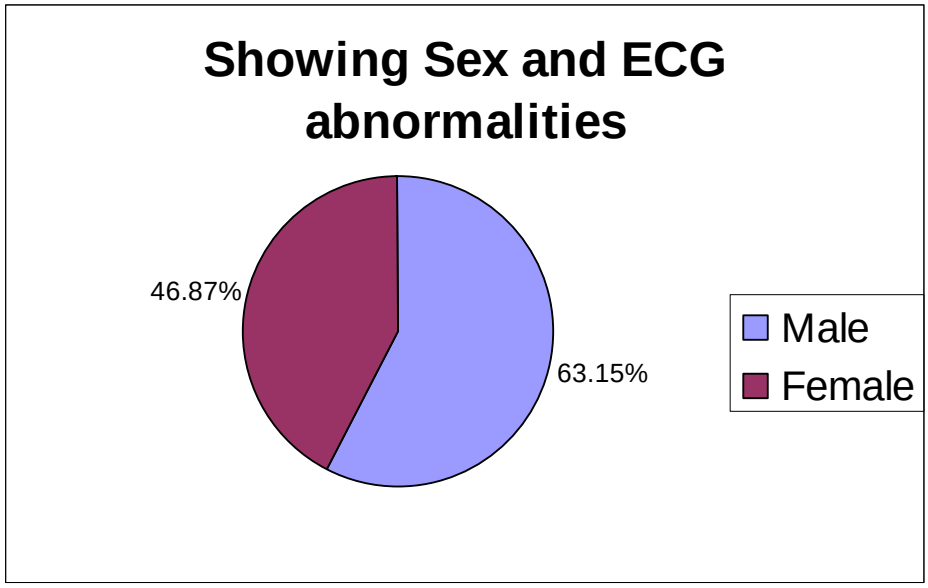
Number of cases in the age group below 20 years were 6(31.57%). The number of cases in the age group of 31-40 were 3(15.78%). One was in the age group of above 70 (5.26%).

Incidence appears more in females than males and the percentage of females in this study was 62.7%.

In females the common age group of poisoning was below 20 years of age and the number of cases in this age group were 14(43.75%). Number of female patients in the age group of 21-30 years were 10(31.25%) and between 31-40 years were 8(25%). It is shown in table 2 given below.

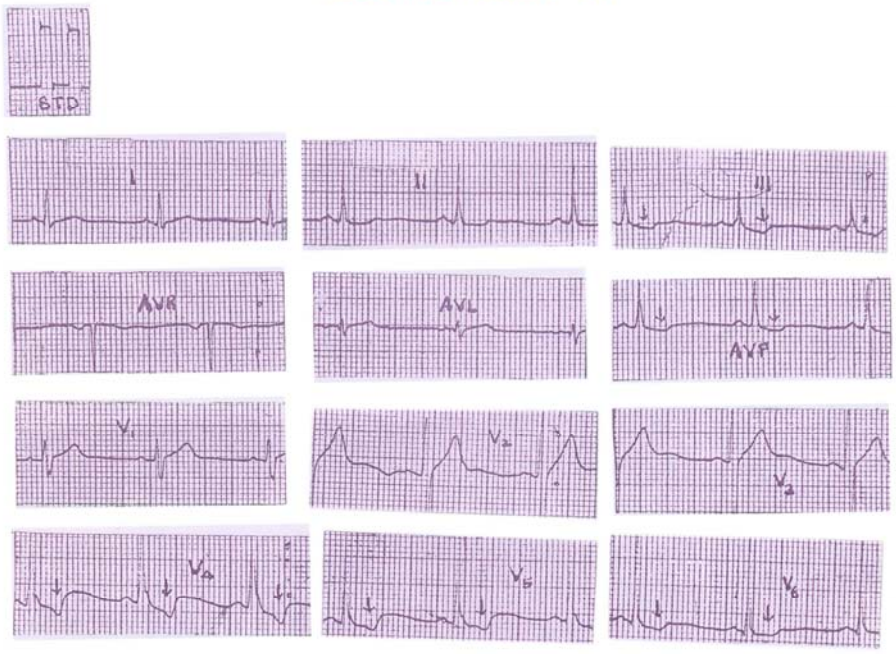
Tab 2. showing sex incidence

Age Group in Years	Male		Female	
	Number of Patients	%	Number of Patients	%
20 and below	6	31.57%	14	43.75%
21 – 30	9	47.36%	10	31.25%
31 – 40	3	15.78%	8	25%
Above 70	1	5.26%		
Total	19	37.2%	32	62.7%


Showing Sex and ECG abnormalities
46.87%
63.15%
Male
Female

CASE NUMBER - 5

ECG NUMBER -III



Electrocardiogram showing ST - DEPRESSION in L_{III} , AVF, V_{4-6}

High incidence in female sex was below 20 yrs and in the male, 20 – 30 yrs of age.

MORTALITY AND AGE

Overall mortality in this study was 1(1.96%). Patient was a female in the age group of 20 – 30.

Table 3. showing age in relation to mortality

AGE GROUP IN YEARS	NUMBER OF PATIENTS	PERCENTAGE
20 - 30	1	1.96%
Total	1	1.96%

High mortality could be due to delay in admission and consumption of more number of seeds. The death was due to ventricular fibrillation.

MORTALITY AND SEX

The percentage of mortality in male was NIL and in female was 1.96% this is shown in table 4 given below.

CASE NUMBER - 7

ECG NUMBER - II

Electrocardiogram showing SINUS BRADYCARDIA - RR interval of 1.32 seconds representing a rate of 45 per minute with normal P,QRS - T complex.

Tab 4. Showing sex in relation to mortality

Age Group in Years	Male		Female	
	Number of Patients	%	Number of Patients	%
1(1.96%)	Nil	-	1	1.96%

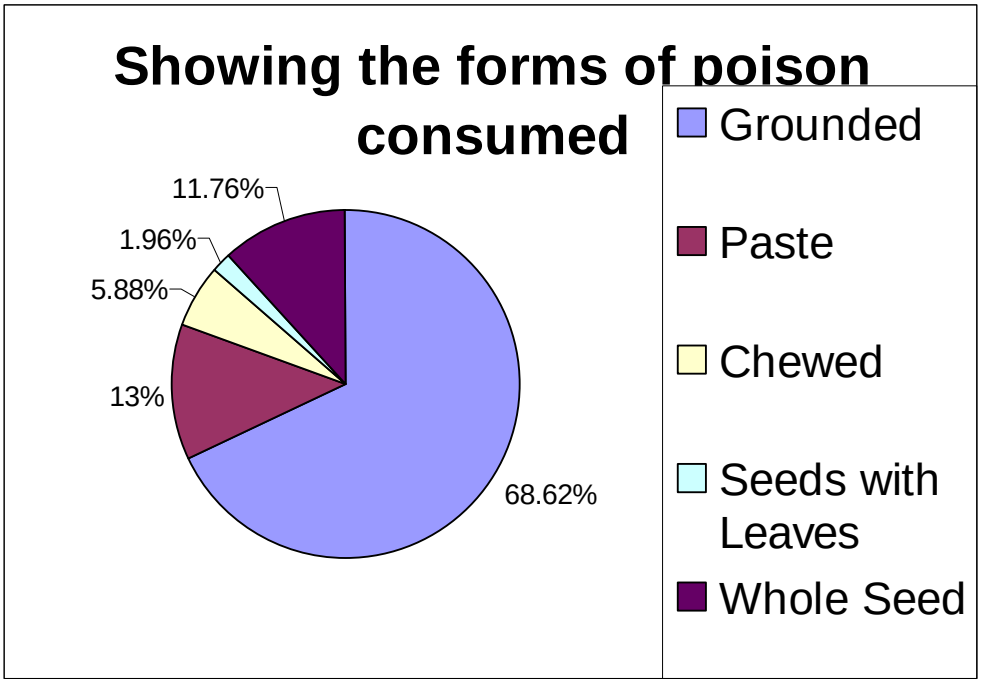
FORMS OF CONSUMPTION

The seeds were taken in many forms but the most common form of consumption was grounded form.

Number of patients consumed in grounded form were 35 (68.62%) and in paste form were 7(13%) and in bitten and chewed form were 3(5.88%). Only 1(1.96%) patient had consumed leaves along with seeds. It is shown in table 5.

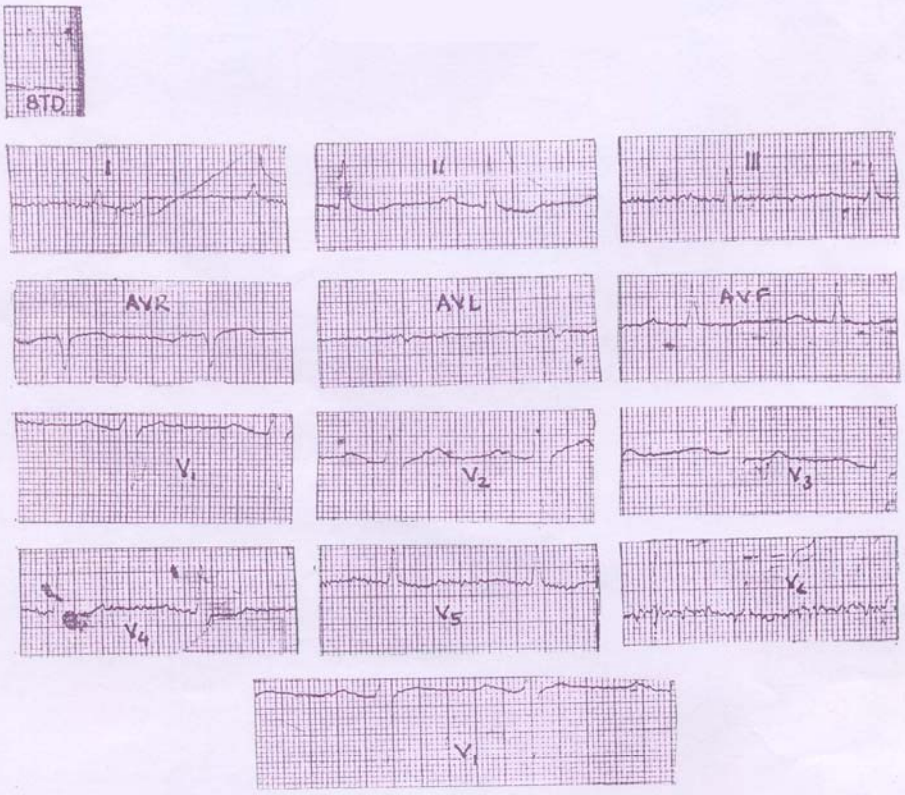
Tab 5. Showing the forms of poison consumed.

Total Number Of Patients	Form of Consumption	Number of Patients	Percentage
51	Grounded	35	68.62%
	Paste	7	13%
	Chewed	3	5.88%
	Seeds with Leaves	1	1.96%
	Whole seed	6	11.76%


Showing the forms of poison consumed
11.76%
1.96%
5.88%
13%
68.62%
Grounded
Paste
Chewed
Seeds with Leaves
Whole Seed

CASE NUMBER - 9

ECG NUMBER - 1II



Electrocardiogram showing 1.SINUS BRADYCARDIA - rate 53/minute, RR interval of 1.08 seconds 2. FIRST DEGREE ATRIO VENTRICULAR BLOCK evidenced by prolonged PR - interval of 0.36 seconds

In grounded and paste form the alkaloid availability is more and hence the electrocardiographic manifestations and mortality were high in this form.

Most of the poisons consumed were seeds, poisoning by leaves were less. Even in those cases they did not exhibit serious manifestations.

In the patient who died, the form of consumption was grounded form.

TIME INTERVAL BETWEEN CONSUMPTION

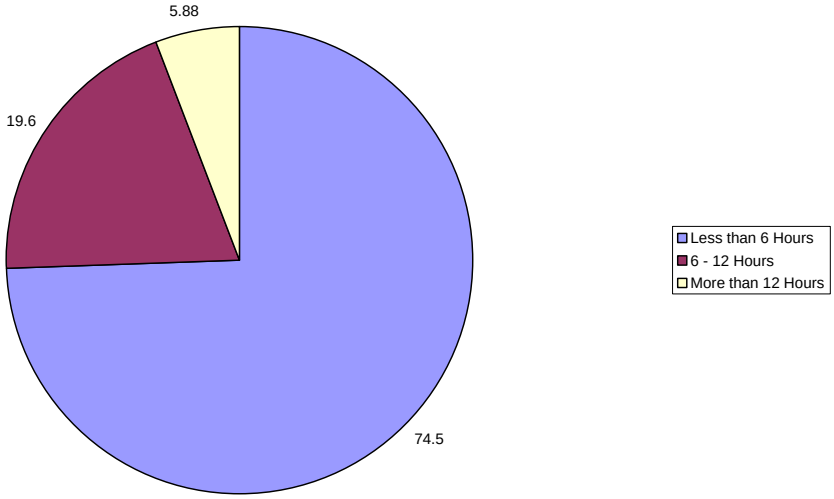
OF POISON AND ADMISSION

Number of patients admitted within 6 hours of poisoning were 38(74.5%) between 6 – 12 hours of poisoning were 10(19.6%) and beyond 12 hour of poisoning were 3(5.88%). This is shown in table 6 given below.

Tab 6. Showing time of admission after poisoning

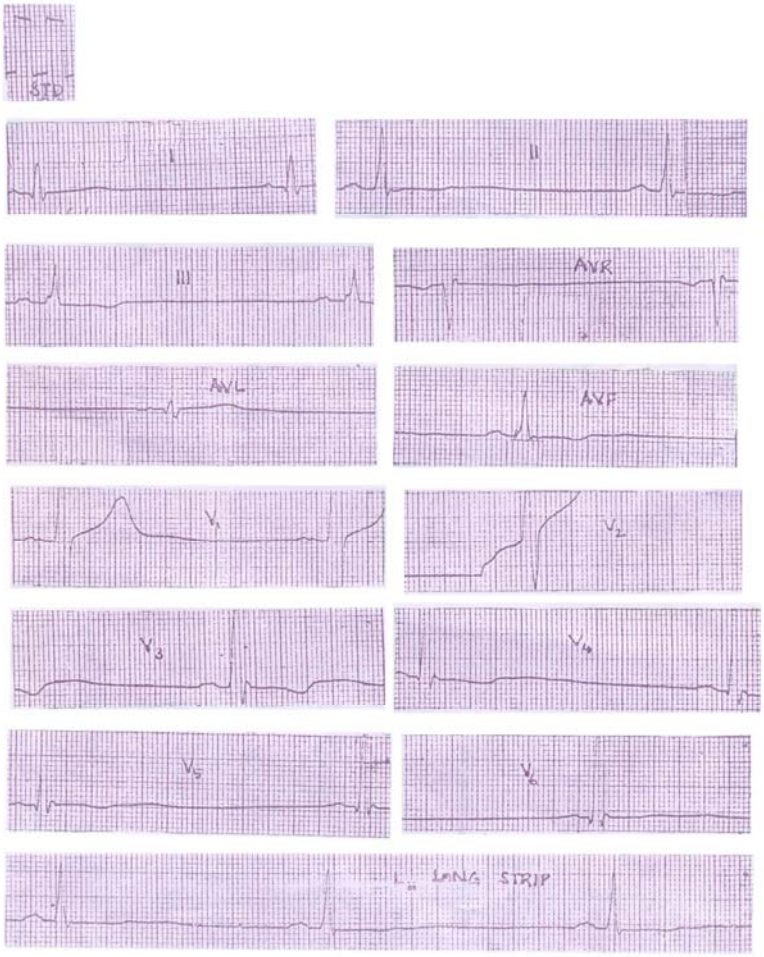
Total Number of Patients	Time Interval between poisoning and admission	Number of patients	Percentage
53	Less than 6 hours	38	74.5%
	6 – 12 hours	10	19.6%
	More than 12 hours	3	5.88%

Showing Time of admission after poisoning



CASE NUMBER - 13

ECG NUMBER - II



Electrocardiogram showing FIRST DEGREE ATRIOVENTRICULAR BLOCK as evidenced by prolonged PR interval of 0.28 sec., rate 27/minute

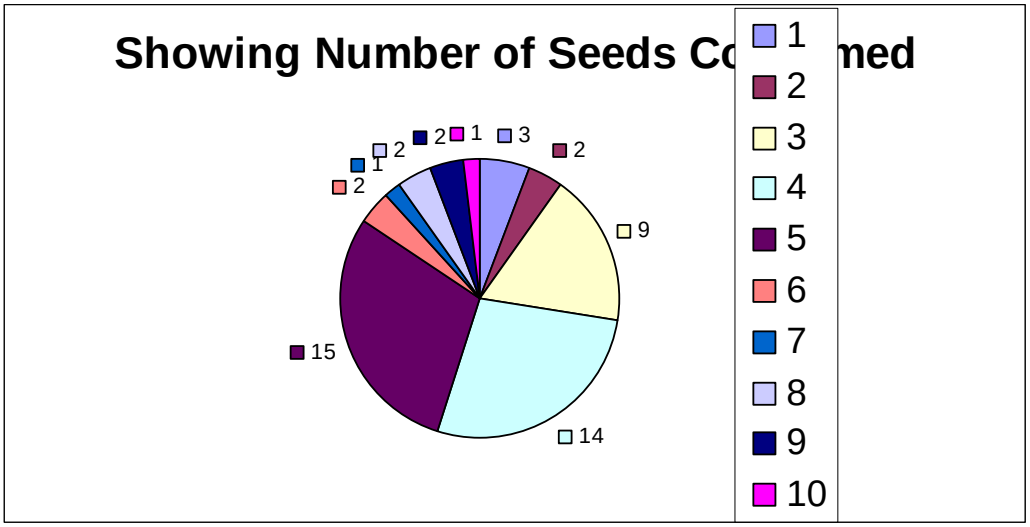
When the delay was more than 10 – 12 hours the chance of mortality was high.

NUMBER OF SEEDS CONSUMED

Number of patients consumed 1 seed were 3(5.88%), 2 seeds were 2(3.92%), 3 seeds were 9(17.64%), 4 seeds were 14(27.45%), 5 seeds were 15(29.41%), 6 seeds were 2(3.92%), 11 seeds were 1(1.96%). This is shown in table 7 given below.

Tab 7: Showing number of seeds consumed

Number of Seeds Consumed	Number of Patients	Percentage
1	3	5.88%
2	2	3.92%
3	9	17.64%
4	14	27.45%
5	15	29.41%
6	2	3.92%
7	1	1.96%
8	2	3.92%
9	-	-
10	2	3.92%
11	1	1.96%
Total Number of Patients	51	


Showing Number of Seeds Co med
1
2
3
4
5
6
7
8
9
10
3
2
9
14
15
2
1
2
2
1

CASE NUMBER-19

ECG NUMBER-1V

Electrocardiogram showing SINUS BRADYCARDIA - rate 45/minute, RR interval 1.36 seconds and normal P,QRS, and T complex.

Mortality appears high when patient consumed more than 3 seeds and also the form of consumption being grounded or paste.

There seems to be a definite abnormality in ECG, even though there is no mortality, if patient had taken even one seed.

The ECG changes and mortality had more relationship with the grounded, chewed or paste form of seeds rather than the number of seeds.

AUTOPSY

In one case we were able to do a complete postmortem and harvest heart fully. Tissues were taken at various places of conducting system and detailed histopathological examination was done.

Macroscopic appearance of various organs especially heart were studied. Stomach and duodenum were congested, stomach had showed fragments of seeds. There was engorgement of veins of the heart.

INCIDENCE OF ECG ABNORMALITIES

Out of the total of 51 patients, 27 patients had electrocardiographic abnormalities and 24 patients had normal ECGs. This is shown in table 8 given below.

CASE NUMBER - 27

ECG NUMBER - III

Electrocardiogram showing 1.SINUS TACHYCARDIA - rate 102/minute 2.SECOND DEGREE ATRIOVENTRICULAR BLOCK IN AVR as evidenced by the second 'P' wave is not followed by QRS complex. PR interval is 0.12 second in all leads.

Tab 8: showing incidence of ECG abnormalities

Total	Number of cases with ECG abnormalities	%	Number of cases with Normal ECG	%
51	27	52.94%	24	47.05%

Out of 24 normal cases, 2(3.92%) patient had consumed only one seed. 2(3.92%) patients had consumed 2 seeds, 8(15.68%) patients had consumed 3 seeds, 8(15.68%) patients had consumed 4 seeds and 2(3.92%) patients had taken 5 seeds and the 1(1.96%) patient had taken 8 seeds, 1(1.96%) patient had taken 10 seeds.

In general ECG abnormalities were noted when patients consumed more than 3 seeds.

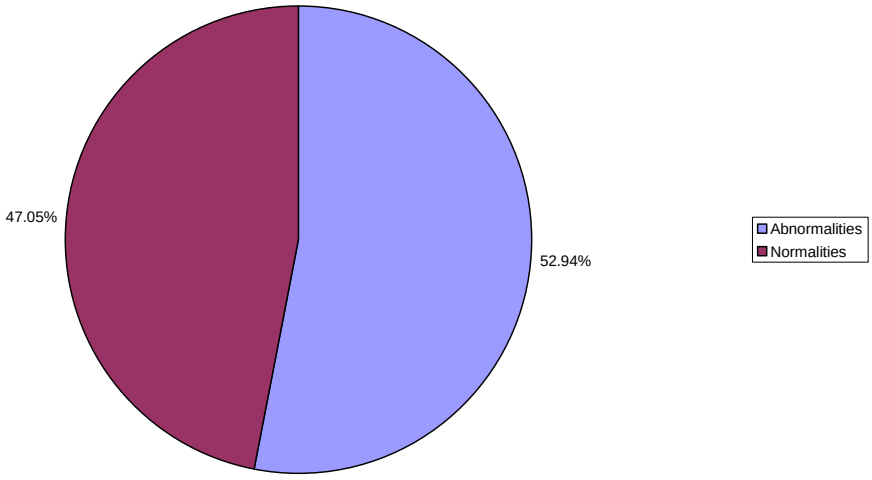
Approximately 52.94% of cases of oleander seed poisoning had ECG abnormalities. According to another study done by BoseTK, Basu RK in India[8], ECG abnormalities occurred in 46% of cases.

AGE AND ECG ABNORMAILITIES

Out of the total of 20 patients admitted in the age group below 20 years (60%) patients had showed ECG abnormalities.

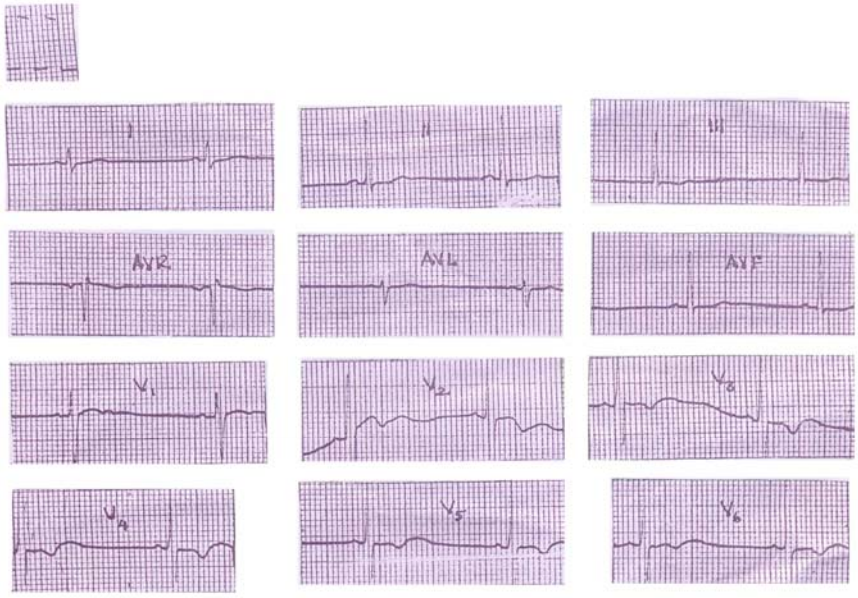
Out of 19 patients in the age group 21 – 30 years 10(52.63%) patients had developed ECG abnormalities.

Showing incidence of ECG abnormalities



CASE NUMBER - 33

ECG NUMBER - IV



Electrocardiogram showing 1.SINUS BRADYCARDIA - rate 56/minute

2. T WAVE INVERSION in L_{III} and V_{16}.

Out of 11 patients in the age group of 31 – 40 years 6(54.54%) cases had ECG abnormalities. This is shown in table 9 given below.

Tab 9: Showing age and ECG abnormalities.

Age Group In years	No. of Patients	No. of Patients with ECG changes	%
20 and below	20	12	60%
21 – 30	19	10	52.63%
31 – 40	11	6	54.54%

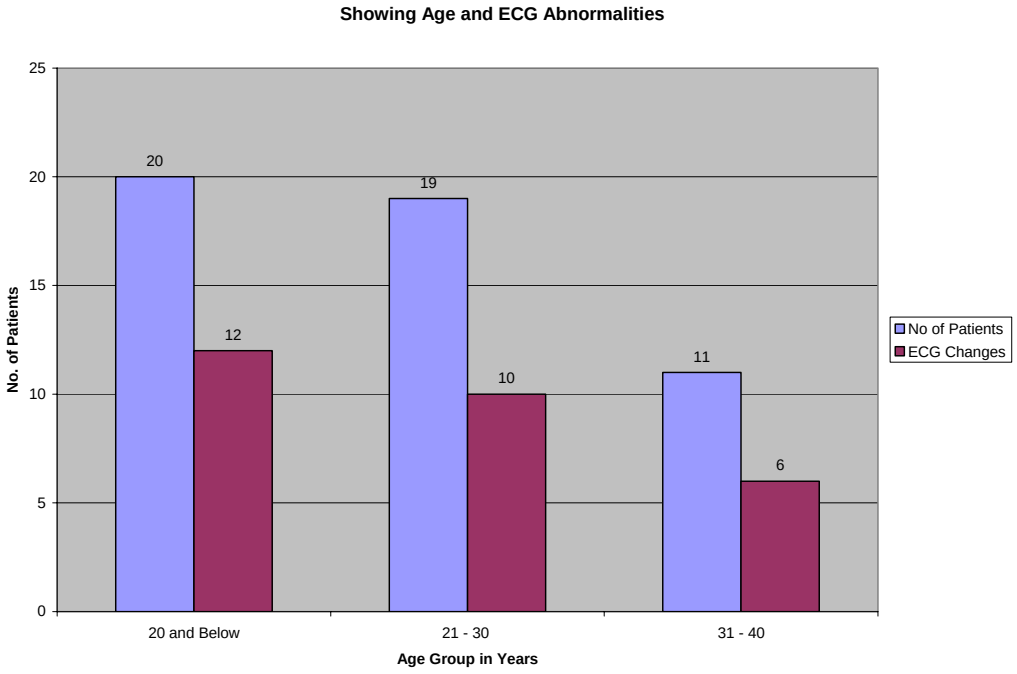
In our study ECG abnormalities were seen equally in all age groups.

SEX AND ECG ABNORMALITIES

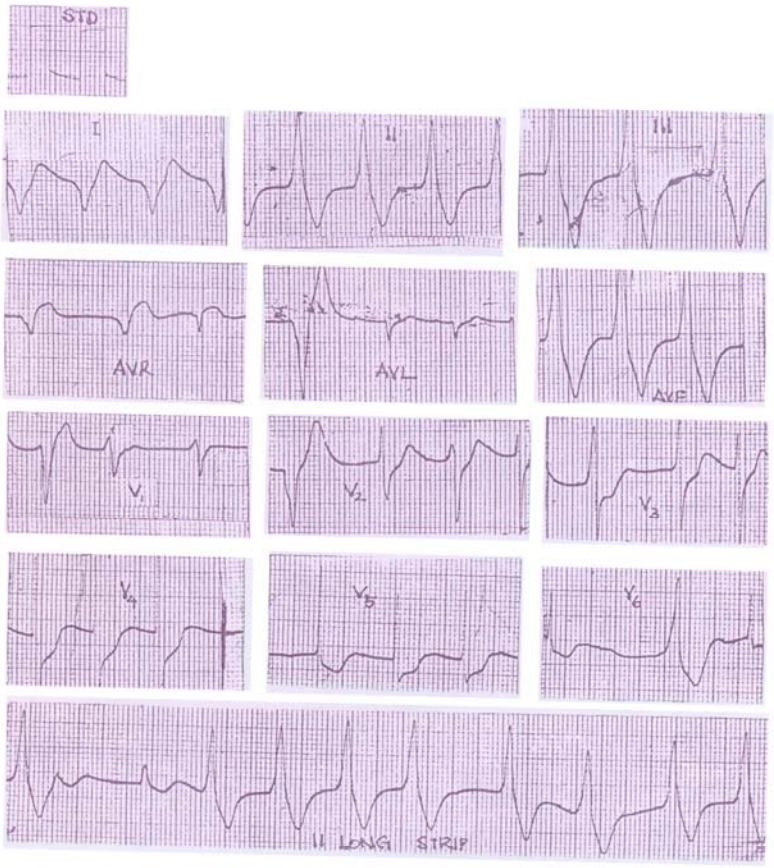
Out of total 19 males, 12(63.15%) males had developed ECG abnormalities and out of 32 females, 15(46.87%) had ECG abnormalities. This is shown in table 10 given below.

Tab 10: Showing sex and ECG abnormalities.

Sex	Total No. of Patients	Patients with ECG changes	Percentage
Male	19	12	63.15%
Female	32	15	46.87%


Showing Age and ECG Abnormalities
25
20
15
10
5
0
No. of Patients
20
12
19
10
11
6
20 and Below
21 - 30
31 - 40
Age Group in Years
No of Patients
ECG Changes

CASE NUMBER - 39
ECG NUMBER - I



Electrocardiogram showing VENTRICULAR TACHYCARDIA (ACCELERATED IDIOVENTRICULAR RHYTHM) evidenced by rate of 106/minute, absent 'P' waves, wide and bizarre QRS complexes and capture beats in AVL.

There is a marginal increase in ECG abnormalities noted in males by 13.15%.

TIME OF APPEARANCE OF ECG ABNORMALITIES

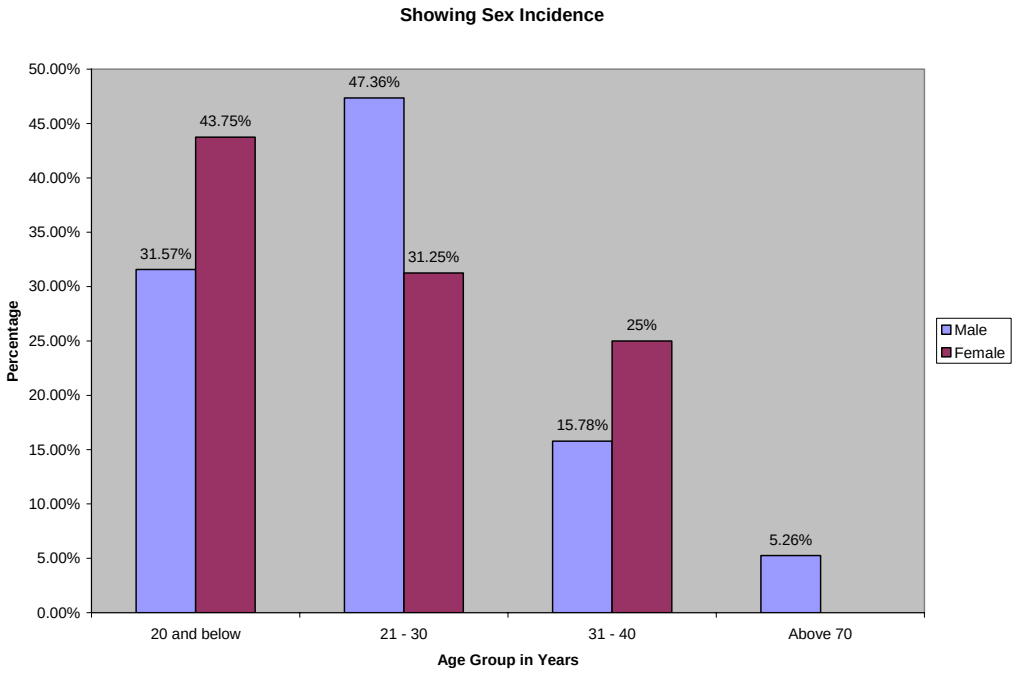
Out of the total of 51 cases 6(83.33%) cases were admitted within 2 hours of poisoning and in that five cases 5(80%) had ECG abnormalities. In one 1(1.96%) patient ECG abnormalities appeared as late as 24 hours after poisoning. In this study ECG abnormalities were noted as early as within 2 hours and as late as 24 hours of poisoning.

NUMBER OF SEEDS AND ECG ABNORMALITIES

In this study out of 51 patients 3(5.88%) patients consumed only one seed. In that 3 cases 1 (33.33%) patient had sinus bradycardia and 2(66.66%) patients had no arrhytmias. ECG abnormalities appear even in one seed.

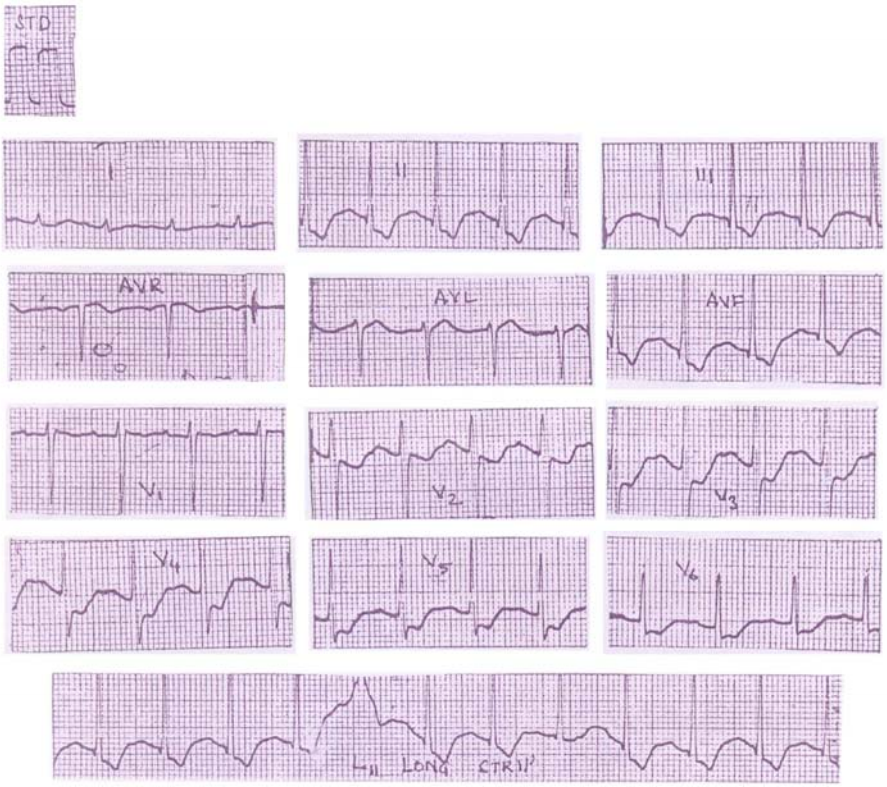
2(3.93%) patients had consumed 2 seeds and both had normal ECGS . 9(17.64%) patients had taken 3 seeds. In that 8(8.88%) of them had no ECG changes. Only one patient had sinus bradycardia. No other changes were noted.

14(27.45%) cases had consumed 4 seeds. In that 14 cases, 6(42.85%) patients had developed arrhythmias in the form of sinus bradycardia, sinoartrial block, first degree atrioventricular block, T wave


Showing Sex Incidence
50.00%
45.00%
40.00%
35.00%
30.00%
25.00%
20.00%
15.00%
10.00%
5.00%
0.00%
Percentage
31.57%
43.75%
47.36%
31.25%
15.78%
25%
5.26%
20 and below
21 - 30
31 - 40
Above 70
Age Group in Years
Male
Female

CASE NUMBER - 45

ECG NUMBER - IV



Electrocardiogram showing SINUS TACHYCARDIA WITH RATE RELATED ST, T CHANGES - RR interval 0.52 second, rate 115/minute, normal P,QRS complex and ST and T wave inversion in L_{II}, L_{III}, AVF, $V_{2\text{-}6}$.

inversion, ST depression and ventricular tachycardia. 8(57.14%) patients had normal ECG.

15(29.41%) patients consumed 5 seeds and 13(86.66%) of them had developed arrhythmias in the form of sinus bradycardia, Sinus tachycardia, SA Block, Ischaemic changes and 2(13.33%) patients had normal ECGs.

2(3.92%) patients had taken 6 seeds and both(100%) of them had showed arrhythmias in the form of sinus bradycardia block.

1(1.96%) patient admitted with consumption of 7 seeds had arrhythmias in the form of sinus bradycardia and sinus tachycardia with second degree AV block.

2(3.93%) cases had taken 8 seeds and arrhythmias were seen in 1(50%) patient one had normal ECG.

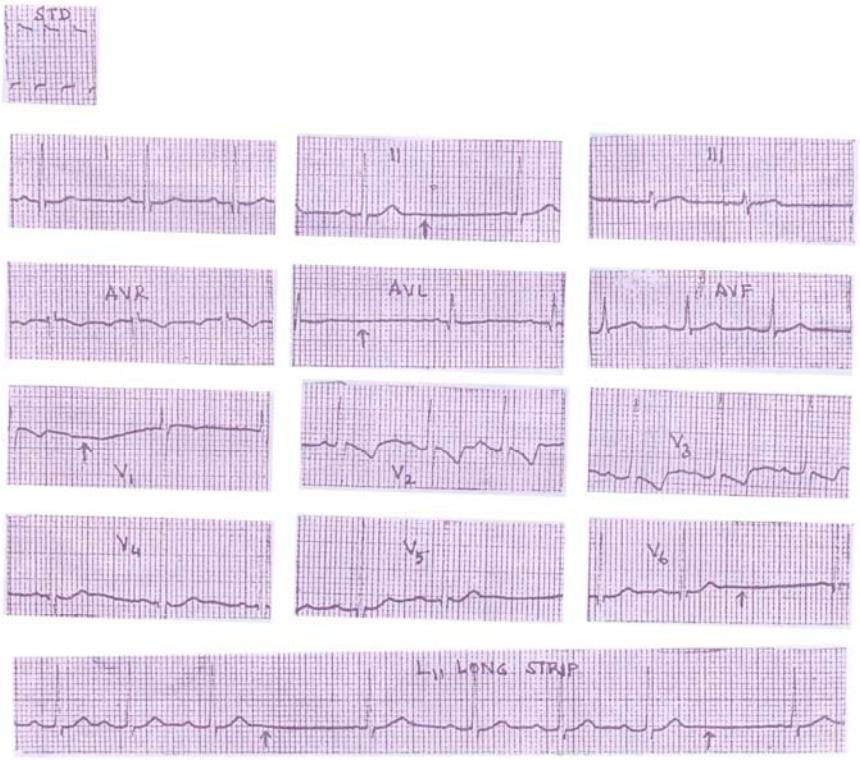
2(3.92%) patients had admitted with history of consumption of 10 seeds. 1 case(50%) had normal ECG and the other had type 1 AV Block and sinus bradycardia.

1 patient had taken 11 seeds and in that patient serial ECGs showed sinus bradycardia, first degree AV Block.

This study shows ECG abnormalities are seen even with one seed. If they consumed more than 5 seeds there is more possibilities for occurence of arrhythmias. This is shown in table 11.

CASE NUMBER - 46

ECG NUMBER - III



Electrocardiogram showing SINOATRIAL BLOCK IN L_{II}, AVI $_4$, V_1, V_4, V_5, V_6, AND L_{11} long strip as evidenced by short and long RR interval. Long RR interval is due to complete omission of one P and QRS complex due to dropped heat.

Table 11. Showing number of seeds taken and ECG Abnormalities

No of seeds taken	Total no of cases	Patients with ECG Abnormalities											
		Abnormal ECGs	%	Sinus Bradycardia	Sinus Tachycardia	ST↓	T↓	SA Block	1^0AV Block	2^0AV Block	VT	Normal ECGs	
												No. of Cases	%
1	3	1	33.33	1	-	-	-	-	-	-	-	2	66.66
2	2	-	-	-	-	-	-	-	-	-	-	2	100
3	9	1	17.64	1	-	-	-	-	-	-	-	8	88.8
4	14	6	42.85	2	-	1	1	1	1	-	1	8	57.14
5	15	13	86.66	7	2	2	1	1	-	-	-	2	13.33
6	2	2	100	2	-	-	-	-	-	-	-	-	-
7	1	1	-	1	1	-	-	-	-	1	-	-	-
8	2	1	50	1	-	-	-	-	-	-	-	1	50
10	2	1	50	-	-	-	-	-	1	-	-	1	50
11	1	1	100	1	-	-	-	-	1	-	-	-	-

VARIOUS FORM OF ECG ABNORMALITES

Out of 27 patients with ECG abnormalities 16(59.25%) patients had sinus bradycardia, 3(11.11%) patients showed sinus tachycardia.

2(7.4%) patients showed T wave inversion and 3(11.11%) patients showed ST segment depression. SA Block was noticed in 2(7.4%) patients.

3(11.11%) patients had showed first degree atrioventricular block, 1(3.7%) patient had second degree atrioventricular block

One patients (3.7%) had ventricular tachycardia.

ECG ABNORMALITY AND MORTALITY

In this study 1 patient expired. ECG showed ventricular Tachycardia.

This is shown in Table 12.

Table 12. Showing various forms of Arrhythmias

ECG Abnormalities	Sinus Bradycardia	Sinus Tachycardia	SA Block	ST Depression	'T' Inversion	1^0 AV Block	2^0 AV Block	Ventricular Tachycardia	Total Abnormal ECGs
Number of Patients	16	3	2	3	2	3	1	1	27
Percentage %	59.25%	11.11%	7.4%	11.11%	7.4%	11.11%	3.7%	3.7%	100%

TIME OF DISAPPEARANCE OF ECG ABNORMALITY

Out of 27 patients with ECG abnormalities 10(37.03%) patients ECG reverted to normal within 24hrs after admission. 8(29.6%) patients ECG became normal between 24 – 48 hours after admission. 7(25.92%) patients ECG became normal between 48 – 72 hours and 1 (3.7%) patients ECG became normal between 72 – 96 hours after admission and 1 (3.7%) patients after 96 hours. This is shown in table 13 given below.

The ECG abnormality persisted upto 5 days so in all cases ECG to be monitored for 5 days compulsorily.

Tab 13. Showing time of disappearance of ECG abnormalities

Time of disappearance of ECG abnormalities in hours	No. of Patients	%	No. of deaths
Less than 24 hours	10	37.03%	-
24 – 48	8	29.6%	-
48 – 72	7	25.92%	1
72 – 96	1	3.7%	-
96 - 120	1	3.7%	-

Yellow Oleander poisoning is common under the age group of below 30 years. Female patients are more in number than male patients.

The cardiac arrythmias in yellow oleander poisoning are associated with high serum cardiac glycosides level and electrolyte disturbances like hyperkalemia[56].

The mean cardiac glycoside level of 2.88 n mol/ litre present with AV Block. The mean cardiac glycoside level of 3.10 n mol / litre present with both AV Block and sinus node dysfunction[56].

Patients with moderate toxicity present with Electrocardiographic changes of prolonged PR interval later proceding to AV dissociation Patients with severe toxicity dies due to ventricular fibrillation[56].

Patients usually have conduction defects affecting sinus node and AV node and a few cases present with atrial and ventricular tachyarrythmias or ventricular ectopic beats that are typical of digoxin poisoning[26].

Studies conducted by Bose TK & Basu RK published in 1990 in Calcutta showed that 82% percentage of patients were females, while it was 62% percentage in our studies.

The number of seeds varied from Half to fifteen in their study, while it was one to Eleven seeds in our study.

Nearly seventy five percentage of patients reached hospital within 6 – 8 hours in both studies.

Patients with symptoms of vomiting in our study was 26.31% which was 30.66% in that study.

Palpitation symptoms were present in 11.76% of our patients, which was 12% in that study.

One patient presented with diarrhea and another patient with syncope in our study. 5(9.8%) patients had hyperkalemia in our study. Patients admitted within 6 hours of poisoning, patients who were given early stomach wash, patients who had early vomiting within few hours after consumption, patients who had taken seeds after food intake had good prognosis and outcome.

Patients were discharged with 100% normal ECG in our studies.

Multiple dose activated charcoal binds cardiac glycosides in the gut lumen and promote their elimination. They reduced the fatalites by about 70% in studies conducted in Srilanka on yellow oleander seed poisoning[19,58].

Antidigoxin fab fragments[37,18] have been found to be effective in reducing the severe cardiac arrythmias and mortality in patients with yellow oleander seed poisoning. The results have been published in journals and studies conducted in **Srilanka**[**42**,20,15,14,27,62,59,2].

CONCLUSION

1. Common age groups of Oleander seed poisoning in our study was less than 30 years.
2. Study revealed higher incidence of poisoning in females.
3. Mortality was related to delay in admission, stomach wash, grounded, chewed or paste from of consumption, electrolyte imbalance like hyperkalemia and cardiac arrythmias.
4. Study revealed ECG abnormalities in about 53% of patients.
5. There was increased mortality if the seed consumption was more than three in number.
6. The ECG abnormalities in males exceeded the females by nearly 20%.
7. ECG abnormalities appeared as early as 2 hrs and as late as 24 hours of poisoning.
8. ECG abnormalities were equal in all age groups studied.
9. The ECG abnormalities had no independent relationship with the number of seeds of consumption.
10. Sinus bradycardia was the commonet arrythmia observed in our study.
11. Ventricular tachycardia and fibrillation were also observed.

12. Patients given early stomach wash with or without activated charcoal had good prognosis and out come.
13. ECG abnormalities lasted for 5 days which emphasizes ECG monitoring or sequential ECGs for minimum of five days.
14. Patients on discharge had 100% normal ECGS.
15. Bundle Branch Block was not noted in this study.
16. Next to Sinus bradycardia, I^{o} AV Block and ST-T changes were noted in this study.
17. Vomiting and palpitation were the common symptoms of presentation.
18. Symptoms of syncope and diarrhea were also reported.

PROFORMA

1. NAME
2. AGE
3. SEX
4. OCCUPATION
5. LITERACY
6. I.P.NUMBER
7. DATE OF ADMISSION
8. DATE OF DISCHARGE
9. DATE AND TIME OF POISONING
10. DATE AND TIME OF ADMISSION
11. TIME INTERVAL BETWEEN CONSUMPTION OF POISON AND ADMISSION
12. DETAILS OF POISONING
    a. COLOUR
    b. NUMBER OF SEEDS
    c. WITH LEAVES
    d. WITHOUT LEAVES
    e. QUANTITY
    f. FORM GROUNDED
    g. CHEWED
    h. PASTE
    i. WITH FOOD
    j. EMPTY STOMACH
13. BEFORE ADMISSION
    a. VOMITING YES / NO
    b. TIME INTERVAL BETWEEN CONSUMPTION & VOMITING YES / NO

c. FIRST AID

14. AFTER ADMISSION

a. GASTROINTESTINAL SYMPTIONS

i. BURNING SENSATION OF MOUTH YES / NO

ii. TINGLING SENSATION OF TONGUE YES / NO

iii. DRYNESS OF THROAT YES / NO

iv. VOMITING YES / NO

v. DIARRHOEA

b. CARDIOVASCULAR SYMPTOMS

i. SYNCOPE YES / NO

ii. PALPITATION YES / NO

iii. DYSPNOEA YES / NO

c. CENTRAL NERVOUS SYSTEM SYMPTIONS

i. BLURRING OF VISION YES / NO

ii. HEADACHE YES / NO

iii. ALTERED SENSORIUM YES / NO

iv. TETANIC CONVULSIONS YES / NO

v. COMA YES / NO

15. CLINICAL EXAMINATION

a. VITAL SIGNS

b. TEMPERATURE

c. PULSE

d. BLOOD PRESSURE

e. RESPIRATORY RATE

f. PUPILS

g. CARDIOVASCULAR SYSTEM

h. RESPIRATORY SYSTEM

i. ABDOMEN

j. CENTRAL NERVOUS SYSTEM

16. INVESTIGATIONS
    a. URINE
        i. ALBUMIN
        ii. SUGAR
        iii. DEPOSITS
    b. BLOOD
        i. UREA
        ii. SUGAR
        iii. SERUM CREATININE
        iv. SERUM ELECTROLYTES'
    c. ELECTROCARDIOGRAM
17. AUTOPSY

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S. No	Name	Age /Sex	IP.No	Date & Time of Admission	Date & Time Poisoning	Time Interval in Hours	S.W with Charcoal	No. Seeds /Form	Symptoms			Electrocardiogram								B S Mgs%	B U Mgs%	S Cr Mgs%	S Na Mg/dl	S K Mg/dl
									V	Pa	O	I	II	III	IV	V	VI	VII	VIII					
1.	Eswari	24/F	884194	3/7/06 6.40pm	3/7/06 2.10pm	4.30	-	5/P				SB	SB	SB	SB	SB	SB	SB	N	92	20	0.8	135	4.2
2.	Seetha	25/F	885513	15/7/06 6.25am	14/7/06 10.15pm	8.10	-	5/G	-	-	-	ST↓	ST↓	N	N	N	N	-	-	108	24	0.8	136	4.0
3.	Anitha	29/F	887013	8/8/06 6.00pm	8/8/06 2.45pm	3.15	+	4/G	-	-	-	N	N	N	N	N	-	-	-	96	30	1.0	135	4.5
4.	Annam	38/F	888003	18/8/06 1.10pm	18/8/06 11.10am	2.00	-	4/G	-	-	-	N	N	N	N	-	-	-	-	112	26	1.0	132	4.5
5.	Rama mirtham	25/F	891016	17/9/06 6.00am	16/8/06 10.00pm	8.00	-	4/G	-	-	-	ST↓	ST↓	ST↓	N	N	N	N	-	110	32	1.0	130	4.2
6.	Lalitha	21/F	891218	20/9/06 11.30am	20/9/06 8.45am	2.45	+	4/G	-	-	-	N	N	N	N	N	-	-	-	130	30	0.8	132	4.0
7.	Jeyapaul	24/M	891312	21/9/06 6.15pm	21/9/06 10.45am	7.30	-	4G	-	-	-	SB	SB	SB	SB	N	N	N	-	129	36	1.0	130	3.8
8.	Rani	22/F	892034	1/10/06 9.40am	1/10/06 8.40am	1.00	-	4G	-	-	-	N	N	N	N	-	-	-	-	115	24	0.8	132	4.0
9.	Vijayakumar	25/M	892939	9/10/06 8.30pm	9/10/06 10.30am	10.00	-	8G	-	-	-	SB I^0AVB	SB I^0AVB	SB I^0AVB	SB	N	N	N	N	118	26	1.0	130	42
10	Ilayaraja	21/M	894539	25/10/06 12.30am	24/10/06 10.00pm	2.30	+	3P	-	-	-	N	N	N	N	-	-	-	-	100	30	1.0	136	4.0
11	Kolajinathan	28/M	895942	8/11/06 10.10pm	8/11/06 5.25pm	4.45	-	5G	+	-	-	N	N	N	N	N	-	-	-	122	28	1.0	132	4.2
12.	Raji	26/F	896239	11/11/06 12.30pm	11/11/06 7.20am	5.10	+	4P	-	-	-	N	N	N	N	N	-	-	-	108	24	1.0	130	3.5
13	Murugesan	32/M	896450	13/11/06 8.25pm	13/11/06 3.25pm	4.50	-	11G	-	-	-	SB I^0AVB	SB I^0AVB	N	N	N	-	-	-	112	28	1.2	130	5.5
14	Kokila	15/F	918451	3/12/06 11.30am	13/12/06 5.00am	6.30	-	4P	-	+	-	SB	SB	SB	SB	N	N	N	-	126	30	1.0	128	4.5
15	Joyce mary	16/F	918469	3/12/06 4.10pm	3/12/06 7.10pm	3.00	-	8/G	+	-	-	N	N	N	N	N	N	-	-	130	20	1.0	130	4.4
16	Malarvizhi	19/F	918654	4/12/06 5.05pm	4/12/06 12.15pm	4.50	-	3/W	-	-	-	N	N	N	N	N	N	-	-	132	26	1.0	128	4.0
17	Kalaiyarasu	17/M	919093	9/12/06 3.00pm	9/12/06 6.40am	8.30	+	5/G	-	-	-	SB	SB	SB	SB	N	N	-	-	106	28	1.0	130	4.0
18	Malika	15/F	919506	13/12/06 7.50pm	13/12/06 2.50pm	5.00	-	5/G	-	-	-	SB	SB	N	N	N	N	-	-	112	30	1.0	132	5.2
19	Savithri	35/F	920632	23/12/06 8.20pm	23/12/06 5.00pm	3.20	-	4/P	-	-	+	SB	SB	SB	SB	N	N	-	-	118	28	0.8	128	4.5
20	Sathiyaraj	20/M	921120	28/12/06 2.40am	27/12/06 10.00pm	4.40	-	5/P	+	-	-	ST	ST	ST	ST	N	N	N	N	108	30	0.8	130	4.2
21	Ramesh	26/M	921246	29/12/06 8.50pm	29/12/06 3.15pm	5.35	+	3/G	-	-	-	N	N	N	N	N	-	-	-	96	20	1.0	128	4.0
22	Parvathi	33/F	921887	05/01/07 1.40am	4/1/07 10.00pm	3.40	-	3G	+	-	-	N	N	N	N	N	-	-	-	100	20	1.0	130	4.0
23	Malarvizhi	15/F	921939	6/1/07 12.15pm	6/1/07 6.15am	6.00	-	5/W	+	-	-	N	N	N	N	-	-	-	-	98	26	1.0	132	4.0
24	Subhiah	17/M	922800	15/01/07 3.50pm	15/01/07 10.00am	5.50	-	2/G	-	-	-	N	N	N	N	-	-	-	-	102	20	0.8	130	4.2
25	Ranjitha	14/F	922945	23/1/07 9.10pm	23/1/07 4.00pm	5.10	-	4/G	+	-	-	N	N	N	N	-	-	-	-	108	24	1.0	130	4.5

26	Chitra	29/F	923328	27/1/07 8.15pm	27/1/07 4.15pm	3.30	+	4/G	-	-	-	N	N	N	N	-	-	-	-	132	30	1.0	128	4.0
27	Vanakamudi	35/M	923419	28/1/07 10.30am	27/1/07 10.00pm	12.30	-	7/G	+	-	+	2^0AV Block	2^0AV Block	ST II ^{0}AVB	ST	N	-	-	-	130	20	1.0	130	5.5
28	Muniappan	18/M	928092	4/2/07 9.10pm	4/2/07 5.10pm	4.00	-	3/G	+	-	-	N	N	N	N	-	-	-	-	100	20	0.8	130	4.0
29	Suresh	24/M	928708	13/2/07 10.15pm	13/2/07 6.00pm	4.15	-	1/W	-	-	-	N	N	N	N	N	-	-	-	86	20	0.8	136	4.2
30	Parvathi	35/F	928710	13/2/07 7.50pm	13/2/07 2.00pm	5.50	-	2/G	-	-		N	N	N	N	N	-	-	-	86	20	0.8	130	4.0
31	Laxmi	32/F	930109	19/2/07 11.30pm	19/2/07 6.15pm	5.15	-	5/P	+	+		SB	SB	SB	N	N	N	N	-	90	30	1.0	135	4.8
32	Padmini	35/F	930519	22/2/07 7.15pm	22/2/07 2.30pm	4.45	+	3/W	-	-		N	N	N	N	-	-	-	-	96	28	0.8	130	4.0
33	Revathi	32/F	931752	5/3/07 9.45pm	5/3/07 5.15pm	4.30	-	5/G	-	+		SB	SB	SB T↓	SB T↓	N	N	N	N	100	30	1.0	130	4.9
34	Vasu	20/M	933125	17/03.07 3.00am	16/03/07 10.00pm	5.00	-	5/G	-	-		SB	SB	SB	SB	SB	N	N	-	112	28	0.8	135	4.0
35	Radhika	37/F	933152	18/3/07 9.30pm	18/3/07 4.30pm	5.00	-	5/G	-	-		SAB	SAB	N	N	N	-	-	-	90	26	0.8	129	4.0
36	Thirumani	35/M	934953	29/3/07 4.40pm	29/3/07 1.00pm	3.40	-	3/G	+	-		SB	SB	N	N	N	N	-	-	102	36	1.1	130	4.2
37	Vinodhini	17/F	935153	1/4/07 11.40pm	1/4/07 7.30am	4.10	+	3/G	+	-		N	N	N	N	N	-	-	-	108	20	0.8	129	3.5
38	Anitha	18/F	935561	5/4/07 2.00pm	5/4/07 10.00am	4.00	-	3Ch	+	-		N	N	N	N	N	-	-	-	110	24	1.0	135	3.9
39	Vembu	26/F	935575	6/4/07 6.10pm	6/4/07 6.10am	12.00	-	4/G	-	+		VT	-	-	-	-	-	-	-	120	30	1.1	135	5.5
40	Malaiyappan	27/M	939757	28/4/07 12.45am	27/4/07 8.00pm	4.45	+	6/G	-	-		SB	SB	N	N	N	-	-	-	86	22	1.0	130	3.8
41	Sumathi	27/F	940069	11/5/07 5.10pm	11/5/07 1.10pm	4.00	-	10/G	+	-		N	N	N	N	N	-	-	-	100	32	1.0	128	4.0
42	Parthiban	18/M	940106	12/5/07 3.40pm	12/507 7.30am	8.10	-	5/G	-	-		SB	SB	SB	N	N	N	-	-	132	28	0.8	134	4.2
43	Suresh	26/M	942633	21/5/07 11.20pm	21/5/07 7.30pm	3.50	-	4/W	-	-		N	N	N	N	N	-	-	-	118	32	1.0	130	4.5
44	Chinnaraja	20/M	943445	29/5/07 9.00pm	29/5/07 7.00pm	2.00	-	5/G	-	-	-	T↓	T↓	T↓	N	N	N	-	-	108	30	1.0	132	5.6
45	Rekha	18/F	945012	10/6/07 8.55pm	10/6/07 2.10pm	6.45	-	5/P	+	-	-	SB	SB	SB	ST	ST	N	N	-	110	32	1.0	128	4.0
46	Bama	19/F	945249	13/6/07 4.15am	12/6/07 10.15pm	6.00	+	1 Ch	-	-	-	SAB	SAB	SAB	N	N	N	N	-	96	22	0.8	130	4.2
47	Usha	19/F	945716	17/6/07 11.00am	17/6/07 9.00am	2.00	-	5/G	-	+	-	SB	SB	SB	SB	N	N	N	-	102	30	1.0	134	40
48	Muthu	22/M	945828	18/06/07 3.00pm	18/06/07 9.30am	5.30	-	10/G	+	-	-	I ^{0}AVB	I ^{0}AVB	I ^{0}AVB	N	N	N	-	-	132	24	0.8	132	4.2
49	Sankari	18/F	946536	24/06/07 2.45am	23/06/07 11.00pm	3.45	-	1/W	-	-	-	N	N	N	N	N	N	-	-	92	22	0.8	128	4.0
50	Nithiya	19/F	947182	29/06/07 3.15pm	29/06/07 7.45am	7.30	-	5/G	-	+	-	T↓	T↓	T↓	N	N	N	N	-	128	32	1.0	135	4.5
51	Rajeswari	19/F	947199	30/06/07 11.10pm	30.06.07 11.00am	12.10	-	4/G	-	-	-	S T↓	S T↓	S T↓	N	N	N	N	-	112	20	0.8	129	5.4

ECG MASTER CHART

S. No.	Name	Ip.No.	ECG No	Rate/Rhythm Per/minute	Axis	'P' Wave	PR in Sec	QRS	'T'	STSEG	QTC	'U'	Impression
1.	Eswari	884194	I	56/Mt Sinus	N	N	0.165	N	N	N	0.38	N	Sinus Bradycardia
			II	49/Mt Sinus	N	N	0.16	N	N	N	0.35	N	Sinus Bradycardia
			III	52/Mt Sinus	N	N	0.16	N	N	N	0.35	N	Sinus Bradycardia
			IV	58/Mt Sinus	N	N	0.16	N	N	N	0.38	N	Sinus Bradycardia
			V	60/Mt Sinus	N	N	0.16	N	N	N	0.38	N	Sinus Bradycardia
			VI	60/Mt Sinus	N	N	0.16	N	N	N	0.38	N	Sinus Bradycardia
			VII	60/Mt Sinus	N	N	0.16	N	N	N	0.38	N	Sinus Bradycardia
			VIII	72/Mt Sinus	N	N	0.16	N	N	N	0.43	N	Normal
2.	Seetha	885513	I	78/Mt Sinus	N	N	0.12	N	N	↓L_{II} L_{III} Avf	0.48	N	Ischemia
			II	78/Mt Sinus	N	N	0.12	N	N	↓L_{II} L_{III} Avf	0.42	N	Normal
			III	82/Mt Sinus	N	N	0.12	N	N	N	0.42	N	Normal
			IV	84/Mt Sinus	N	N	0.12	N	N	N	0.42	N	Normal
			V	84/Mt Sinus	N	N	0.12	N	N	N	0.42	N	Normal
3.	Anitha	887013	I	88/Mt Sinus	N	N	0.12	N	N	N	0.42	N	Normal
			II	82/Mt Sinus	N	N	0.12	N	N	N	0.42	N	Normal
			III	90/Mt Sinus	N	N	0.12	N	N	N	0.43	N	Normal
			IV	88/Mt Sinus	N	N	0.12	N	N	N	0.42	N	Normal
			V	88/Mt Sinus	N	N	0.12	N	N	N	0.42	N	Normal
4.	Annam	888003	I	90/Mt Sinus	N	N	0.16	N	N	N	0.44	N	Normal
			II	88/Mt Sinus	N	N	0.14	N	N	N	0.42	N	Normal
			III	92/Mt Sinus	N	N	0.12	N	N	N	0.42	N	Normal
			IV	86/Mt Sinus	N	N	0.14	N	N	N	0.44	N	Normal
			V	88/Mt Sinus	N	N	0.14	N	N	N	0.42	N	Normal
5.	Rama mirtha m	891016	I	76/Mt Sinus	N	N	0.12	N	ST↓ L_3 v_4 – V_6	↓AVF V_{4-6}	0.43	N	ST↓
			II	78/Mt Sinus	N	N	0.12	N	ST↓ L_3 AVF	V_{4-6}	0.43	N	ST↓
			III	71/Mt Sinus	N	N	0.12	N	ST↓ L_3 v_4 – V_6	↓AVF V_{4-6}	0.43	N	ST↓
			IV	78/Mt Sinus	N	N	0.12	N	N	N	0.42	N	Normal
			V	76/Mt Sinus	N	N	0.12	N	N	N	0.42	N	Normal
			VI	79/Mt Sinus	N	N	0.12	N	N	N	0.42	N	Normal
			VII	80/Mt Sinus	N	N	0.12	N	N	N	0.43	N	Normal
6.	Lalitha	891218	I	80/Mt Sinus	N	N	0.12	N	N	N	0.44	N	Normal
			II	78/Mt Sinus	N	N	0.14	N	N	N	0.44	N	Normal
			III	88/Mt Sinus	N	N	0.12	N	N	N	0.44	N	Normal
			IV	86/Mt Sinus	N	N	0.12	N	N	N	0.44	N	Normal
7.	Jeyapaul	891312	I	58/Mt Sinus	N	N	0.16	N	N	N	0.44	N	SB
			II	54/Mt Sinus	N	N	0.16	N	N	N	0.43	N	SB
			III	52/Mt Sinus	N	N	0.16	N	N	N	0.44	N	SB
			IV	72/Mt Sinus	N	N	0.12	N	N	N	0.42	N	Normal
			V	78/Mt Sinus	N	N	0.12	N	N	N	0.44	N	Normal
			VI	80/Mt Sinus	N	N	0.14	N	N	N	0.42	N	Normal
			VII	78/Mt Sinus	N	N	0.12	N	N	N	0.42	N	Normal
			VIII	86/Mt Sinus	N	N	0.12	N	N	N	0.42	N	Normal
8.	Rani	892034	I	78/Mt Sinus	N	N	0.12	N	N	N	0.42	N	Normal
			II	80/Mt Sinus	N	N	0.12	N	N	N	0.42	N	Normal
			III	82/Mt Sinus	N	N	0.12	N	N	N	0.43	N	Normal
			IV	86/Mt Sinus	N	N	0.12	N	N	N	0.42	N	Normal

9.	Vijaya kumar	892939	I	58/Mt Sinus	N	N	0.36	N	N	N	0.40	N	SB I ^{0}AVB
			II	56/Mt Sinus	N	N	0.32	N	N	N	0.40	N	SB with I ^{0}AVB
			III	53/Mt Sinus	N	N	0.36	N	N	N	0.36	N	Normal
			IV	68/Mt Sinus	N	N	0.20	N	N	N	0.42	N	Normal
			V	72/Mt Sinus	N	N	0.16	N	N	N	0.44	N	Normal
			VI	78/Mt Sinus	N	N	0.16	N	N	N	0.43	N	Normal
			VII	78/Mt Sinus	N	N	0.16	N	N	N	0.44	N	Normal
10	Ilayaraja	894539	I	78/Mt Sinus	N	N	0.12	N	N	N	0.42	N	Normal
			II	80/Mt Sinus	N	N	0.14	N	N	N	0.42	N	Normal
			III	76/Mt Sinus	N	N	0.12	N	N	N	0.43	N	Normal
			IV	82/Mt Sinus	N	N	0.12	N	N	N	0.42	N	Normal
11	Kolajinathan	895942	I	81/Mt Sinus	N	N	0.12	N	N	N	0.45	N	Normal
			II	79/Mt Sinus	N	N	0.14	N	N	N	0.42	N	Normal
			III	82/Mt Sinus	N	N	0.12	N	N	N	0.44	N	Normal
			IV	84/Mt Sinus	N	N	0.14	N	N	N	0.42	N	Normal
12.	Raji	896239	I	82/Mt Sinus	N	N	0.16	N	N	N	0.40	N	Normal
			II	81/Mt Sinus	N	N	0.16	N	N	N	0.41	N	Normal
			III	78/Mt Sinus	N	N	0.12	N	N	N	0.40	N	Normal
			IV	83/Mt Sinus	N	N	0.16	N	N	N	0.40	N	Normal
			V	84/Mt Sinus	N	N	0.16	N	N	N	0.40	N	Normal
13	Murugesan	896450	I	52/Mt Sinus	N	N	0.24	N	N	N	0.42	N	SB with I ^{0}AVB
			II	27/Mt Sinus	N	N	0.28	N	N	N	0.40	N	SB with I ^{0}AVB
			III	52/Mt Sinus	N	N	0.24	N	N	N	0.42	N	SB with I ^{0}AVB
			IV	72/Mt Sinus	N	N	0.20	N	N	N	0.44	N	Normal
			V	76/Mt Sinus	N	N	0.20	N	N	N	0.44	N	Normal
			VI	88/Mt Sinus	N	N	0.16	N	N	N	0.45	N	Normal
			VII	86/Mt Sinus	N	N	0.12	N	N	N	0.44	N	Normal
14	Kokila	918451	I	58/Mt Sinus	N	N	0.14	N	N	N	0.45	N	Normal
			II	60/Mt Sinus	N	N	0.12	N	N	N	0.44	N	Normal
			III	59/Mt Sinus	N	N	0.12	N	N	N	0.40	N	SB
			IV	60/Mt Sinus	N	N	0.14	N	N	N	0.40	N	SB
			V	72/Mt Sinus	N	N	0.14	N	N	N	0.41	N	SB
			VI	78/Mt Sinus	N	N	0.12	N	N	N	0.42	N	Normal
			VII	82/Mt Sinus	N	N	0.12	N	N	N	0.44	N	Normal
			VIII	80/Mt Sinus	N	N	0.12	N	N	N	0.44	N	Normal
15	Joyce mary	918469	I	78/Mt Sinus	N	N	0.16	N	N	N	0.40	N	Normal
			II	84/Mt Sinus	N	N	0.16	N	N	N	0.42	N	Normal
			III	83/Mt Sinus	N	N	0.16	N	N	N	0.42	N	Normal
			IV	86/Mt Sinus	N	N	0.12	N	N	N	0.40	N	Normal
			V	92/Mt Sinus	N	N	0.12	N	N	N	0.41	N	Normal
16	Malarvizhi	918654	I	82/Mt Sinus	N	N	0.16	N	N	N	038	N	Normal
			II	86/Mt Sinus	N	N	0.16	N	N	N	0.40	N	Normal
			III	83/Mt Sinus	N	N	0.16	N	N	N	0.40	N	Normal
			IV	82/Mt Sinus	N	N	0.16	N	N	N	0.40	N	Normal
			V	81/Mt Sinus	N	N	0.16	N	N	N	0.42	N	Normal
17	Kalaiyarasu	919093	I	58/Mt Sinus	N	N	0.12	N	N	N	0.36	N	SB
			II	52/Mt Sinus	N	N	0.12	N	N	N	0.38	N	SB
			III	47/Mt Sinus	N	N	0.12	N	N	N	0.39	N	SB
			IV	52/Mt Sinus	N	N	0.12	N	N	N	0.40	N	SB
			V	68/Mt Sinus	N	N	0.12	N	N	N	0.42	N	Normal
			VI	72/Mt Sinus	N	N	0.12	N	N	N	0.40	N	Normal
			VII	78/Mt Sinus	N	N	0.12	N	N	N	0.40	N	Normal
			VIII	80/Mt Sinus	N	N	0.12	N	N	N	0.40	N	Normal
18	Malika	919506	I	52/Mt Sinus	N	N	0.14	N	N	N	0.40	N	SB
			II	58/Mt Sinus	N	N	0.14	N	N	N	0.40	N	SB
			III	68/Mt Sinus	N	N	0.16	N	N	N	0.44	N	Normal
			IV	72/Mt Sinus	N	N	0.12	N	N	N	0.42	N	Normal
			V	88/Mt Sinus	N	N	0.12	N	N	N	0.44	N	Normal
			VI	82/Mt Sinus	N	N	0.12	N	N	N	0.44	N	Normal
19	Savithri	920632	I	48/Mt Sinus	N	N	0.16	N	N	N	0.36	N	SB
			II	52/Mt Sinus	N	N	0.16	N	N	N	0.36	N	SB

			III	52/Mt Sinus	N	N	0.16	N	N	N	0.36	N	SB
			IV	46/Mt Sinus	N	N	0.16	N	N	N	0.36	N	SB
			V	72/Mt Sinus	N	N	0.14	N	N	N	0.40	N	Normal
			VI	78/Mt Sinus	N	N	0.12	N	N	N	0.42	N	Normal
			VII	80/Mt Sinus	N	N	0.12	N	N	N	0.40	N	Normal
20	Sathiyaraj	921120	I	100/MtSinus	N	N	0.16	N	N	N	0.50	N	ST
			II	102/MtSinus	N	N	0.16	N	N	N	0.50	N	ST
			III	100/MtSinus	N	N	0.16	N	N	N	0.50	N	ST
			IV	120/MtSinus	N	N	0.20	N	N	N	0.56	N	ST
			V	96/Mt Sinus	N	N	0.12	N	N	N	0.45	N	Normal
			VI	92/Mt Sinus	N	N	0.12	N	N	N	0.44	N	Normal
			VII	97/Mt Sinus	N	N	0.12	N	N	N	0.44	N	Normal
			VIII	90/Mt Sinus	N	N	0.12	N	N	N	0.44	N	Normal
21	Ramesh	921246	I	90/Mt Sinus	N	N	0.14	N	N	N	0.44	N	Normal
			II	88/Mt Sinus	N	N	0.12	N	N	N	0.42	N	Normal
			III	92/Mt Sinus	N	N	0.12	N	N	N	0.44	N	Normal
			IV	92/Mt Sinus	N	N	0.12	N	N	N	0.44	N	Normal
			V	91/Mt Sinus	N	N	0.12	N	N	N	0.44	N	Normal
22	Parvathi	921887	I	91/Mt Sinus	N	N	0.12	N	N	N	0.40	N	Normal
			II	86/Mt Sinus	N	N	0.14	N	N	N	0.42	N	Normal
			III	82/Mt Sinus	N	N	0.12	N	N	N	0.44	N	Normal
			IV	88/Mt Sinus	N	N	0.12	N	N	N	0.40	N	Normal
			V	89/Mt Sinus	N	N	0.14	N	N	N	0.40	N	Normal
23	Malarvizhi	921939	I	72/Mt Sinus	N	N	0.17	N	N	N	0.40	N	Normal
			II	76/Mt Sinus	N	N	0.12	N	N	N	0.42	N	Normal
			III	80/Mt Sinus	N	N	0.14	N	N	N	0.44	N	Normal
			IV	82/Mt Sinus	N	N	0.12	N	N	N	0.44	N	Normal
			V	80/Mt Sinus	N	N	0.12	N	N	N	0.44	N	Normal
24	Subhiah	922800	I	73/Mt Sinus	N	N	0.16	N	N	N	0.40	N	Normal
			II	72/Mt Sinus	N	N	0.16	N	N	N	0.41	N	Normal
			III	78/Mt Sinus	N	N	0.12	N	N	N	0.42	N	Normal
			IV	89/Mt Sinus	N	N	0.12	N	N	N	0.44	N	Normal
			V	86/Mt Sinus	N	N	0.12	N	N	N	0.44	N	Normal
25	Ranjitha	922945	I	82/Mt Sinus	N	N	0.16	N	N	N	0.40	N	Normal
			II	88/Mt Sinus	N	N	0.16	N	N	N	0.42	N	Normal
			III	92/Mt Sinus	N	N	0.14	N	N	N	0.40	N	Normal
			IV	88/Mt Sinus	N	N	0.14	N	N	N	0.40	N	Normal
			V	88/Mt Sinus	N	N	0.14	N	N	N	0.40	N	Normal
26	Chitra	923328	I	82/Mt Sinus	N	N	0.16	N	N	N	0.38	N	Normal
			II	81/Mt Sinus	N	N	0.16	N	N	N	0.40	N	Normal
			III	88/Mt Sinus	N	N	0.16	N	N	N	0.40	N	Normal
			IV	89/Mt Sinus	N	N	0.16	N	N	N	0.42	N	Normal
			V	92/Mt Sinus	N	N	0.16	N	N	N	0.40	N	Normal
27	Vanakamudi	923419	I	92/Mt Sinus	N	N	0.14	N	N	N	0.48	N	II ^{0}AVB
			II	98/Mt Sinus	N	N	0.14	N	N	N	0.48	N	II ^{0}AVB
			III	102/MtSinus	N	N	0.12	N	N	N	0.50	N	ST with II ^{0}AVB
			IV	98/Mt Sinus	N	N	0.12	N	N	N	0.44	N	Normal
			V	92/Mt Sinus	N	N	0.12	N	N	N	0.46	N	Normal
			VI	91/Mt Sinus	N	N	0.12	N	N	N	0.46	N	Normal
			VII	92/Mt Sinus	N	N	0.12	N	N	N	0.46	N	Normal
28	Muniappan	928092	I	82/Mt Sinus	N	N	0.16	N	N	N	0.40	N	Normal
			II	84/Mt Sinus	N	N	0.16	N	N	N	0.42	N	Normal
			III	88/Mt Sinus	N	N	0.16	N	N	N	0.42	N	Normal
			IV	87/Mt Sinus	N	N	0.16	N	N	N	0.42	N	Normal
			V	86/Mt Sinus	N	N	0.16	N	N	N	0.42	N	Normal
29	Suresh	928708	I	88/Mt Sinus	N	N	0.12	N	N	N	0.42	N	Normal
			II	89/Mt Sinus	N	N	0.12	N	N	N	0.42	N	Normal
			III	91/Mt Sinus	N	N	0.12	N	N	N	0.40	N	Normal
			IV	82/Mt Sinus	N	N	0.12	N	N	N	0.40	N	Normal
			V	88/Mt Sinus	N	N	0.12	N	N	N	0.40	N	Normal
30	Parvathi	928710	I	78/Mt Sinus	N	N	0.16	N	N	N	0.42	N	Normal
			II	85/Mt Sinus	N	N	0.12	N	N	N	0.44	N	Normal
			III	86/Mt Sinus	N	N	0.12	N	N	N	0.42	N	Normal
			IV	88/Mt Sinus	N	N	0.12	N	N	N	0.44	N	Normal
			V	87/Mt Sinus	N	N	0.12	N	N	N	0.44	N	Normal

31	Laxmi	930109	I	52/Mt Sinus	N	N	0.16	N	N	N	0.48	N	SB
			II	58/Mt Sinus	N	N	0.16	N	N	N	0.46	N	SB
			III	60/Mt Sinus	N	N	0.16	N	N	N	0.46	N	SB
			IV	72/Mt Sinus	N	N	0.12	N	N	N	0.44	N	Normal
			V	78/Mt Sinus	N	N	0.14	N	N	N	0.44	N	Normal
			VI	76/Mt Sinus	N	N	0.12	N	N	N	0.42	N	Normal
			VII	82/Mt Sinus	N	N	0.12	N	N	N	0.42	N	Normal
32	Padmini	930519	I	78/Mt Sinus	N	N	0.12	N	N	N	0.44	N	Normal
			II	81/Mt Sinus	N	N	0.12	N	N	N	0.42	N	Normal
			III	80/Mt Sinus	N	N	0.12	N	N	N	0.43	N	Normal
			IV	89/Mt Sinus	N	N	0.12	N	N	N	0.40	N	Normal
			V	81/Mt Sinus	N	N	0.12	N	N	N	0.42	N	Normal
33	Revathi	931752	I	60/Mt Sinus	N	N	0.14	N	N	N	0.40	N	SB
			II	58/Mt Sinus	N	N	0.14	N	N	N	0.42	N	SB
			III	58/Mt Sinus	N	N	0.16	N	$T\downarrow L_{III}$ V_{1-6}	N	0.40	N	SB With T Inversion
			IV	56/Mt Sinus	N	N	0.16	N	$T\downarrow L3$ V_{1-6}	N	0.41	N	SB With T Inversion
			V	68/Mt Sinus	N	N	0.16	N	N	N	0.40	N	Normal
			VI	72/Mt Sinus	N	N	0.16	N	N	N	0.42	N	Normal
			VII	72/Mt Sinus	N	N	0.16	N	N	N	0.44	N	Normal
			VIII	88/Mt Sinus	N	N	0.12	N	N	N	0.44	N	Normal
34	Vasu	933125	I	59/Mt Sinus	N	N	0.14	N	N	N	0.42	N	SB
			II	58/Mt Sinus	N	N	0.16	N	N	N	0.41	N	SB
			III	60/Mt Sinus	N	N	0.16	N	N	N	0.42	N	SB
			IV	60/Mt Sinus	N	N	0.16	N	N	N	0.42	N	SB
			V	60/Mt Sinus	N	N	0.16	N	N	N	0.44	N	SB
			VI	68/Mt Sinus	N	N	0.12	N	N	N	0.43	N	Normal
			VII	78/Mt Sinus	N	N	0.12	N	N	N	0.44	N	Normal
			VIII	82/Mt Sinus	N	N	0.12	N	N	N	0.44	N	Normal
			IX	81/Mt Sinus	N	N	0.12	N	N	N	0.44	N	Normal
35	Radhika	933152	I	80/Mt Sinus	N	N	0.16	N	N	N	0.40	N	SAB
			II	82/Mt Sinus	N	N	0.16	N	N	N	0.42	N	SAB
			III	84/Mt Sinus	N	N	0.16	N	N	N	0.40	N	Normal
			IV	86/Mt Sinus	N	N	0.16	N	N	N	0.44	N	Normal
			V	88/Mt Sinus	N	N	0.16	N	N	N	0.44	N	Normal
			VI	87/Mt Sinus	N	N	0.16	N	N	N	0.44	N	Normal
36	Thirumani	934953	I	58/Mt Sinus	N	N	0.12	N	N	N	0.40	N	SB
			II	60/Mt Sinus	N	N	0.16	N	N	N	0.41	N	SB
			III	68/Mt Sinus	N	N	0.12	N	N	N	0.40	N	Normal
			IV	72/Mt Sinus	N	N	0.16	N	N	N	0.44	N	Normal
			V	78/Mt Sinus	N	N	0.12	N	N	N	0.42	N	Normal
			VI	79/Mt Sinus	N	N	0.12	N	N	N	0.44	N	Normal
			VII	78/Mt Sinus	N	N	0.12	N	N	N	0.44	N	Normal
37	Vinodhini	935153	I	78/Mt Sinus	N	N	0.12	N	N	N	0.42	N	Normal
			II	76/Mt Sinus	N	N	0.14	N	N	N	0.43	N	Normal
			III	78/Mt Sinus	N	N	0.14	N	N	N	0.42	N	Normal
			IV	82/Mt Sinus	N	N	0.12	N	N	N	0.42	N	Normal
			V	80/Mt Sinus	N	N	0.12	N	N	N	0.42	N	Normal
38	Anitha	935561	I	72/Mt Sinus	N	N	0.12	N	N	N	0.38	N	Normal
			II	79/Mt Sinus	N	N	0.14	N	N	N	0.40	N	Normal
			III	78/Mt Sinus	N	N	0.14	N	N	N	0.38	N	Normal
			IV	82/Mt Sinus	N	N	0.14	N	N	N	0.39	N	Normal
			V	82/Mt Sinus	N	N	0.12	N	N	N	0.40	N	Normal
39	Vembu	935575	I	106 Ventricular	-	Absent	-	Abnormal	Abnormal	-	Absent	N	IDIO Ventricular Tachycardia
40	Malaiyappan	939757	I	59/Mt Sinus	N	N	0.14	N	N	N	0.42	N	SB
			II	60/Mt Sinus	N	N	0.14	N	N	N	0.42	N	SB
			III	72/Mt Sinus	N	N	0.16	N	N	N	0.44	N	Normal
			IV	80/Mt Sinus	N	N	0.12	N	N	N	0.42	N	Normal
			V	80/Mt Sinus	N	N	0.12	N	N	N	0.42	N	Normal
			VI	87/Mt Sinus	N	N	0.12	N	N	N	0.40	N	Normal
41	Sumathi	940069	I	82/Mt Sinus	N	N	0.12	N	N	N	0.40	N	Normal
			II	84/Mt Sinus	N	N	0.14	N	N	N	0.42	N	Normal
			III	83/Mt Sinus	N	N	0.12	N	N	N	0.42	N	Normal

			IV	88/Mt Sinus	N	N	0.12	N	N	N	0.42	N	Normal
			V	87/Mt Sinus	N	N	0.12	N	N	N	0.42	N	Normal
42	Parthiban	940106	I	58/Mt Sinus	N	N	0.16	N	N	N	0.38	N	SB
			II	60/Mt Sinus	N	N	0.16	N	N	N	0.40	N	SB
			III	60/Mt Sinus	N	N	0.12	N	N	N	0.40	N	SB
			IV	68/Mt Sinus	N	N	0.12	N	N	N	0.44	N	Normal
			V	78/Mt Sinus	N	N	0.14	N	N	N	0.44	N	Normal
			VI	75/Mt Sinus	N	N	0.12	N	N	N	0.44	N	Normal
			VII	78/Mt Sinus	N	N	0.12	N	N	N	0.44	N	Normal
43	Suresh	942633	I	78/Mt Sinus	N	N	0.16	N	N	N	0.40	N	Normal
			II	86/Mt Sinus	N	N	0.16	N	N	N	0.40	N	Normal
			III	92/Mt Sinus	N	N	0.16	N	N	N	0.40	N	Normal
			IV	90/Mt Sinus	N	N	0.16	N	N	N	0.40	N	Normal
			V	91/Mt Sinus	N	N	0.16	N	N	N	0.40	N	Normal
44	Chinnaraja	943445	I	82/Mt Sinus	N	N	0.12	N	N	N	0.42	N	N↑↓ Inversion
			II	80/Mt Sinus	N	N	0.12	N	N	N	0.43	N	N↑↓ Inversion
			III	81/Mt Sinus	N	N	0.12	N	N	N	0.42	N	N↑↓ Inversion
			IV	82/Mt Sinus	N	N	0.12	N	N	N	0.44	N	Normal
			V	84/Mt Sinus	N	N	0.12	N	N	N	0.44	N	Normal
			VI	85/Mt Sinus	N	N	0.12	N	N	N	0.42	N	Normal
			VII	81/Mt Sinus	N	N	0.14	N	N	N	0.42	N	Normal
45	Rekha	945012	I	58/Mt Sinus	N	N	0.12	N	N	N	0.40	N	SB
			II	60 /Mt Sinus	N	N	0.12	N	N	N	0.40	N	SB
			III	60 /Mt Sinus	N	N	0.12	N	N	N	0.40	N	SB
			IV	115/Mt Sinus	N	N	0.12	N	↓L_2, V_4-V_6	↓L_2,V_4-V_6	0.56	N	ST with rate related ST –T Changes
			V	108/Mt Sinus	N	N	0.12	N	L_3, V_{24}-V_6	↓V_4-V_6	0.50	N	ST with ST – T Changes
			VI	98/Mt Sinus	N	N	0.14	N	N	N	0.44	N	Normal
			VII	92/Mt Sinus	N	N	0.14	N	N	N	0.40	N	Normal
			VIII	91/Mt Sinus	N	N	0.14	N	N	N	0.42	N	Normal
46	Bama	945249	I	98 /Mt Sinus	N	N	0.12	N	N	N	0.48	N	SAB
			II	98 /Mt Sinus	N	N	0.12	N	N	N	0.48	N	SAB
			III	96 /Mt Sinus	N	N	0.12	N	N	N	0.49	N	SAB
			IV	92 /Mt Sinus	N	N	0.12	N	N	N	0.44	N	Normal
			V	91 /Mt Sinus	N	N	0.12	N	N	N	0.44	N	Normal
			VI	92 /Mt Sinus	N	N	0.12	N	N	N	0.42	N	Normal
			VII	91 /Mt Sinus	N	N	0.12	N	N	N	0.44	N	Normal
47	Usha	945716	I	60 /Mt Sinus	N	N	0.14	N	N	N	0.45	N	SB
			II	60 /Mt Sinus	N	N	0.14	N	N	N	0.44	N	SB
			III	58 /Mt Sinus	N	N	0.12	N	N	N	0.42	N	SB
			IV	60 /Mt Sinus	N	N	0.12	N	N	N	0.40	N	SB
			V	72 /Mt Sinus	N	N	0.12	N	N	N	0.40	N	Normal
			VI	70 /Mt Sinus	N	N	0.12	N	N	N	0.40	N	Normal
			VII	81 /Mt Sinus	N	N	0.12	N	N	N	0.40	N	Normal
48	Muthu	945828	I	78/Mt Sinus	N	N	0.14	N	N	N	0.40	N	Normal
			II	91 /Mt Sinus	N	N	0.12	N	N	N	0.40	N	Normal
			III	88 /Mt Sinus	N	N	0.12	N	N	N	0.41	N	Normal
			IV	87 /Mt Sinus	N	N	0.12	N	N	N	0.40	N	Normal
49	Sankari	946536	I	70 /Mt Sinus	N	N	0.24	N	N	N	0.40	N	I ^{0}AVB

			II	68 /Mt Sinus	N	N	0.24	N	N	N	0.42	N	I ^{0}AVB
			III	71 /Mt Sinus	N	N	0.24	N	N	N	0.40	N	I ^{0}AVB
			IV	72 /Mt Sinus	N	N	0.18	N	N	N	0.41	N	Normal
			V	78 /Mt Sinus	N	N	0.18	N	N	N	0.41	N	Normal
			VI	76 /Mt Sinus	N	N	0.18	N	N	N	0.40	N	Normal
50	Nithiya	947182	I	78 /Mt Sinus	N	N	0.12	N	N	$\downarrow V_3$-V_6	0.42	N	'T' Inversion
			II	76 /Mt Sinus	N	N	0.12	N	N	$\downarrow V_3$-V_6	0.42	N	'T' Inversion
			III	77 /Mt Sinus	N	N	0.12	N	N	$\downarrow V_3$-V_6	0.42	N	'T' Inversion
			IV	78 /Mt Sinus	N	N	0.12	N	N	N	0.43	N	Normal
			V	80 /Mt Sinus	N	N	0.14	N	N	N	0.40	N	Normal
			VI	81 /Mt Sinus	N	N	0.14	N	N	N	0.40	N	Normal
			VII	80 /Mt Sinus	N	N	0.12	N	N	N	0.40	N	Normal
51	Rajes wari	947199	I	72 /Mt Sinus	N	N	0.14	N	N	$\downarrow L_{2,3,}$ avf	0.44	N	ST Depression
			II	78 /Mt Sinus	N	N	0.14	N	N	$\downarrow L_{2,3,}$ avf	0.44	N	ST Depression
			III	80 /Mt Sinus	N	N	0.12	N	N	$\downarrow L_{2,3,}$ avf	0.44	N	ST Depression
			IV	81 /Mt Sinus	N	N	0.12	N	N	N	0.42	N	Normal
			V	80 /Mt Sinus	N	N	0.12	N	N	N	0.40	N	Normal
			VI	82 /Mt Sinus	N	N	0.12	N	N	N	0.40	N	Normal
			VII	81 /Mt Sinus	N	N	0.12	N	N	N	0.40	N	Normal

ABBREVIATIONS

ABBREVIATIONS

S.No.	-	Patient Number
SW	-	Stomach Wash
V	-	Vomiting
Pa	-	Palpitation
O	-	Others
B S	-	Blood Sugar
B U	-	Blood Urea
S C	-	Serum Creatinine
S Na	-	Serum Sodium
S K	-	Serum Potassium
S T	-	Sinus Tachycardia
N	-	Normal
G	-	Grounded
P	-	Paste
Ch	-	Chewed
W	-	Whole
T↓	-	T Inversion
I^0AVB	-	First Degree AtrioVentricular Block
II^0AVB	-	Second Degree AtrioVentricular Block
VT	-	Ventricular Tachycardia
ST↓	-	ST Depression
SAB	-	Sino Atrial Block
Axis	-	Normal (+40 to +60)
SB	-	Sinus Bradycardia